MELLENDICK, KEVAN M., Ph.D. Diet Quality and Cardiovascular Disease Risks in Adolescents. (2016)
Directed by Dr. Cheryl Lovelady. 147 pp.

Obesity and cardiovascular disease (CVD) risk among adolescents have become major public health concerns. Obesity rate has reached 18.4% among 12-19 year olds, and 20.3% of this age group has at least one abnormal blood lipid level. Among obese adolescents, dyslipidemia rate is troubling, at 42.9%. Diet quality may play a key role in the development of obesity and other CVD risks among this age group. Consistently poor diet quality has been observed among adolescents, and research indicates dietary patterns from this age persist into adulthood. Moreover, minimal research has explored connections between diet quality and CVD risk in adolescents. Therefore, the primary aims of this dissertation were: 1) to determine differences in diet quality between obese and non-obese, hypertensive and normotensive, and dyslipidemic and non-dyslipidemic 16 year-olds; 2) to examine relationships between diet quality and CVD risks; 3) to identify relationships between dietary components and CVD risk; and 4) to examine interactions between diet quality and adiposity on CVD risks.

The first study identified greater sweetened beverage intake among obese adolescents. Additionally, fruit and fiber intake were negatively related to body mass index (BMI). Fruit and protein intake were also negatively related to waist circumference (WC). The second study found positive relationships between blood pressure (BP) and BMI and WC. Greater consumption of vegetable, fruit, whole fruit, greens and beans

fiber, and magnesium, and lower consumption of energy from fat were related to decreases in BP.

The second study also showed greater intake of sweetened beverages, and lower intake of omega 3 fatty acids and fiber among participants with elevated total cholesterol (TC). Diet quality, omega 3 fatty acids, fiber, vegetable, and greens and beans were positively related to TC. Lastly, low density lipoprotein was negatively related to vegetable and greens and beans intake. Significant moderation effects by diet quality were observed on the relationships between BMI and systolic BP, and BMI and triglycerides.

These findings implicate elevated BMI, WC, and sweetened beverage intake with greater CVD risk. By contrast, these findings suggest cardio-protective effects for greater vegetable, fruit, and fiber consumption.

DIET QUALITY AND CARDIOVASCULAR DISEASE RISKS IN ADOLESCENTS

by

Kevan M. Mellendick

A Dissertation Submitted to
the Faculty of The Graduate School at
The University of North Carolina at Greensboro
in Partial Fulfillment
of the Requirements for the Degree
Doctor of Philosophy

Greensboro
2016

Approved by

Cheryl Lovelady, PhD, MPH, RD
Committee Chair

For Amanda, the beautiful buttress of all my efforts, and the love of my life.

For Jeanie, my little bear, who makes me smile every day, and gives me the strength to do anything.

For Nana, who always wanted me to be the first “doctor” in the family.

For my parents, who had genuine confidence I could do anything.

For my little brother, whose perception of me is something to which I continue to aspire.

For Debbie & Keith, Kris & Chris, Angie & Glenn, and all of my wonderful, encouraging family.

APPROVAL PAGE

This dissertation written by KEVAN M. MELLENDICK has been approved by the following committee of the Faculty of The Graduate School at The University of North Carolina at Greensboro.

Committee Chair _____

Committee Members _____

_____

_____

_____
Date of Acceptance by Committee

_____
Date of Final Oral Examination

ACKNOWLEDGEMENTS

I must first thank Dr. Cheryl Lovelady for her assistance both in this project and my development as an academic. I came to her unsure whether to even continue this track, and left a significantly better person, let alone researcher or teacher. Her guidance, patience, flexibility, and incomparable talent were genuinely treasured and humbling.

For all her guidance and inputs in this research and my development as a teacher, I would like to thank Dr. Laurie Wideman. Whether in the form of data analysis, lecture development, testing, grading, or general advice, her imprint on my career and life will always reflect her kind nature and unrelenting work ethic.

I would also like to thank Dr. Lenka Shriver and Dr. Lauren Haldeman. They have provided invaluable input into this work, and are role models of professional excellence, conviviality, and family life balance.

Special thanks must be given to James Janssen, the project coordinator without whom all of the RIGHT Track Health projects would surely and quickly stall. He was always quick to provide superb assistance with a friendly disposition. In a few years, he will certainly write a stellar dissertation himself.

This study was funded by the Eunice Kennedy Shriver National Institute of Child Health & Human Development of the National Institutes of Health under award number R01HD078346 [PI; Shanahan] and by NIH award number DK56350 to the Nutrition Obesity Research Center (NORC) at the University of North Carolina Chapel Hill . The

historic data collection was funded by NIMH 55625, NIMH 55584 & NIMH 58144 [PI; Calkins].

TABLE OF CONTENTS

LIST OF TABLES

Page

LIST OF FIGURES

Page

CHAPTER I

INTRODUCTION

As obesity rates in the United States peak among children and adolescents, diet quality continues to be poor. While 18.4% of U.S. adolescents, aged 12-19 years, are now considered obese (1), diet quality measurements consistently reveal well below optimal scores (2–5). Additionally, longitudinal research indicates dietary patterns from adolescence may persist into adulthood (6). Even within this young age group, dyslipidemia is emerging, with approximately 20.3% of US 12-19 year olds having at least one abnormal lipid level (7). Dyslipidemia is even more pronounced among obese adolescents, as 42.9% of that group has at least one abnormal lipid value. Taken altogether, these numbers reflect an increasing concern for the development of chronic diseases, such as cardiovascular disease (CVD), at a young age. With prior work identifying signs of CVD in teenagers (8), the adolescent age group seems to be an ideal target population for prevention efforts and identifying key CVD risks.

As early as 1952, autopsy studies of deceased youth have shown significant development of early cardiovascular disease at very young age. In particular, atherosclerotic lesions, fatty streaks, and fibrous plaques in the coronary arteries are present well before the age of 20, with some present prior to age 10 (8–11). In fact, Berenson et al. found fibrous plaques present in coronary arteries of in approximately 35% of the 204 16-20 year old cadavers examined (8). Based on these studies,

cardiovascular disease should be considered progressive, and originating in adolescence or even earlier.

With increasing prevalence of overweight and obesity among the adolescent population (1), identifying risk for chronic disease among younger age groups has become a priority in preventive medicine and public health efforts. Considering tissue dissection and plaque evaluation is not a viable screening tool for cardiovascular disease intervention, factors related to development and progression of cardiovascular diseases will be examined to identify risk for CVD. Of particular interest are measures of adiposity, blood pressure (BP), and blood lipids.

Overweight and obesity among adolescents are related to alterations in lipid and glucose metabolism, and blood pressure regulation. In conjunction, these alterations represent a greatly increased risk for the metabolic syndrome, which is noted to correspond with chronic systemic inflammation. This inflammation, although considered stable, corresponds to atherosclerotic vascular changes, such as those discovered by Berenson et al (8). However, with appropriate medical and/or lifestyle intervention, these vascular changes are predominantly reversible at this stage of life (12,13). As such, elevated adiposity, hypertension, and chronic inflammation are important indicators of a potentially reversible atherosclerotic process in the adolescent age group. Additionally, obesity, with its known contributions to CVD development and progression, can be considered both a CVD risk and predictor of additional CVD risk.

Still, in order prevent development of these CVD risk indicators, the contributing antecedents must be better understood. The influence of poor lifestyle habits, particularly diet, has been studied thoroughly among the adult population. However, the impact of poor diet on CVD risk in adolescence is not well established. Moreover, very limited data exists connecting measures of whole diet to CVD risk among the younger population.

Specific aspects of nutrition are considered contributors to CVD. Most research focuses on single nutrients (such as sodium and dietary fiber), clusters of nutrients (such as fats and simple sugars), and/or food groups/clusters of foods (such as fruits/vegetables or fast foods) (14–18). However, researchers have noted that use of a validated overall diet quality measure would be preferable in future research, as it can simultaneously encompass multiple aspects of diet quality that may confound or offset the impact of specific nutrients or food groups. The Healthy Eating Index-2010, is one of the validated measures of total diet quality, based on the current Dietary Guidelines for Americans (19).

While most CVD risk research has focused on middle to older age, this study explored potential CVD risk factors in adolescence, a period in which early/initial stages of CVD may manifest. Due to relatively recent increases in known contributing factors to CVD initiation and proliferation, adolescence has emerged as a life stage carrying multiple CVD risk. Yet, the relationships among these CVD risks are to date, largely unexplored. The goal of this study was to elucidate the influence of diet quality on cardiovascular disease risks at a young age. This research utilized data from a long-term,

ongoing longitudinal study to investigate a heterogeneous sample of 16 year olds. The specific aims of this study are listed below.

Specific Aim #1: To identify differences in diet quality of adolescents between those who are obese versus non-obese, who have elevated blood pressure versus normal blood pressure, and who have dyslipidemia versus normal blood lipids.

Hypothesis #1: Diet quality will be poorer in obese adolescents, adolescents with elevated blood pressure, and dyslipidemic adolescents.

Specific Aim #2a: To determine the relationships in adolescents between all variables: gender, race, socio-economic status (SES), diet quality, body mass index, waist circumference, blood pressure, and blood lipids.

Hypothesis #2a: Body mass index and waist circumference will be significantly positively correlated. SES will be significantly positively related to diet quality and HDL cholesterol, and negatively related to blood pressure, total cholesterol, LDL cholesterol, and triglycerides. Boys will have significantly poorer diet quality than girls. Non-white race participants will have significantly higher body mass index and blood pressure than white race participants.

Specific Aim #2b: To specifically determine the relationships among diet quality with body mass index, waist circumference, blood pressure, and blood lipids in adolescents.

Hypothesis #2b: Diet quality will be negatively related to body mass index, waist circumference, blood pressure, total cholesterol, LDL cholesterol, and triglycerides, but positively related to HDL cholesterol. Moreover, body mass index and waist circumference will be positively related to blood pressure, total cholesterol, LDL cholesterol, and triglycerides, but negatively to HDL cholesterol.

Specific Aim #3: To examine the interaction of adiposity and diet quality in their relationship with blood pressure, and blood lipids.

Hypothesis #3: Diet quality will moderate the relationships between measures of adiposity and blood pressure, and blood lipids.

Specific Aim #4: To examine the relationships between specific nutrients and classes of foods with CVD risk factors.

Hypothesis #4: Total energy, total fat, saturated fat, refined grain, and empty calorie intake will be significantly positively related to body mass index and waist circumference. Fruit and vegetable, whole grain, and fiber intake will be significantly negatively related to body mass index and waist circumference. Blood pressure will be significantly positively correlated with sodium intake, and significantly negatively correlated with fruit, vegetable, fiber, potassium, magnesium, calcium, and omega-3 fatty acid intake. Total cholesterol, LDL cholesterol, and triglycerides will be significantly positively associated with added sugars, saturated fatty acids, and trans fatty acids, and negatively associated with monounsaturated fatty acids, omega-3 fatty acids, fiber, fruit,

and vegetable consumption. HDL cholesterol will be significantly negatively related to added sugars, saturated fatty acids and trans fatty acids.

The current study examined the relationships between diet quality and CVD risks, and identified potential differences in these relationships among at-risk and low risk (non-obese, normotensive, normal blood lipids) 16 year olds (hereafter referred to in a general term as adolescents). Additionally, this research has identified and explored potential interactions between diet quality and adiposity in predicting multiple CVD risks. This research treated our measures of adiposity as both CVD risks, potentially predicted by diet quality (specific aim #2), and as a predictor of other CVD risk, along with diet quality (specific aim #3). In addition, this research assessed the interaction of diet quality and adiposity in their relationship to other CVD risks. Recent literature suggests increased CVD risk in metabolically unhealthful lean adults, and low risk for metabolically healthful obese individuals (20–22). As such, examining the interaction of measures of adiposity and diet quality in predicting other CVD risks may provide unique insight into the presence (and development) of metabolically healthful obesity and/or metabolically unhealthful leanness.

Further research may link atherosclerotic processes with diet quality, including the combined influences of diet quality with adiposity on the development of cardiovascular disease.

CHAPTER II

REVIEW OF LITERATURE

Cardiovascular Disease Risk in Adolescents

Obesity, associated with excessive adipose tissue, is highly related to the development of cardiovascular disease (23,24). The accumulation of excessive adipose tissue leads to altered blood lipid metabolism, increased blood pressure, and constant low-level inflammation. These changes associated with excess adiposity can lead to atherosclerosis and blood vessel endothelial dysfunction (25–27). As such, obesity is an independent risk factor for cardiovascular disease development and progression.

The prevalence of obesity among adolescents, while not as high as adults, is still a public health concern. Ogden et al reported 33.6% of US adolescents are overweight, with 18.4% obese (1). These results are based on data from the National Health and Nutrition Examination Survey (NHANES), covering 2009-2010, with overweight and obesity based on body mass index for age ($\geq 85^{th}$ percentile overweight; $\geq 95^{th}$ percentile obese). While NHANES data are unique to the USA and adolescents living here, a recent review examined abdominal obesity specifically across many nations, using waist circumference (WC), as the indicator (28). This review found a wide range in abdominal obesity rates, from 3.8% to 51.7% among 10-19 year olds. Among developed nations, a narrower range was reported, 8.7% to 33.2%. The cutoff point for abdominal obesity

diagnosis using WC had no consensus in this review, and 14 different cutoff points were utilized across 1977-2007, with the most common cutoffs being ≥90th percentile of the sample (as proposed by Cook et al (29)), ≥95thpercentile of the sample, ≥90th percentile of the country, or ≥70th percentile of the country.

Research links BMI directly to atherosclerosis, including a study by Berenson et al, who found significant, strong correlation between the presence of fibrous plaques in the coronary arteries and BMI of adolescents (8). Correlations to plaque presence were strong with BMI (r=0.48). A similar effect was found relating carotid intima-media thickness in adulthood (25-37 years) to BMI (OR, 1.25; 95% CI, 1.01-1.54) and systolic blood pressure (OR, 1.36; 95% CI, 1.08-1.72) measured in childhood (4-17 years) (30). In this study, BMI and blood pressure were measured in childhood, and compared to carotid intima-media thickness, a validated surrogate for coronary artery disease risk, of the same population at 20-year follow-up, ages 25-37. Both BMI (and systolic blood pressure in childhood (4-17 years old) were significant predictors of being in the top versus other three quartiles of carotid intima-media thickness in adulthood (25-37 years old). Another cross-sectional study found carotid intima-media thickness related strongly to BMI z-score (r=0.514) among 61 obese and 25 normal-weight 12-18 year olds in northern Pennsylvania (31). Additional research examined 21 obese adolescents, mean age 17.7 years and mean BMI 41.9, and 12 healthy adolescents, mean age 15.1 and mean BMI 20.1. Results from a single session of cardiac resonance imaging showed a strong correlation between BMI and left ventricular extracellular volume fraction (r=0.58), a measure of subclinical alteration in heart tissue (23).

Research also identifies correlations between adiposity measures and other cardiovascular risk factors, such as BP. For example, data from NHANES 2003-2004 also show BMI relates significantly to both total cholesterol and systolic BP in 727 adolescents (age 15-17 years) (32). Additionally, study of 1950 Brazilian children and adolescents showed strong correlations of WC to systolic BP (r=0.449) and diastolic BP (r=0.374) (33). Further research in Brazil found trends relating both BMI and WC to systolic and diastolic BP among 564 children and adolescents 8-17 years of age (34). Both BMI and WC showed reasonably strong correlation to systolic BP (r=0.415 and 0.461, respectively), with slightly weaker correlations to diastolic BP (r=0.376 and 0.433, respectively), and slightly stronger correlations of WC to BP generally.

Blood Lipids and Cardiovascular Disease Risk in Adolescents

A preponderance of evidence supports abnormally high TC, LDL cholesterol, and TG in the blood, or abnormally low HDL cholesterol in the blood as significant independent risks for heart disease (24,25,35–37). To that effect, annual testing for these blood lipids within the adult population has become rote care to monitor the progression of cardiovascular disease in the primary care setting. The American Heart Association (AHA) highlights specific cut-off values for each of these measures (35,37). Considered abnormally high, and therefore at greater risk for heart disease, are TC ≥200 mg/dL, LDL cholesterol ≥130 mg/dL, and TG ≥150 mg/dL. Considered abnormally low and therefore at greater risk for heart disease is HDL ≤40 mg/dL. However, it should be noted that LDL cholesterol <100 mg/dL and HDL cholesterol ≥60 mg/dL are considered optimal.

Among adolescents, less evidence directly supports the impact of elevated blood lipids on atherosclerosis and cardiovascular disease progression. However, the AHA does supply recommendations for optimal and suboptimal blood lipid values among the childhood and adolescent population.

Unfortunately, prevalence of abnormal lipid levels is relatively high within this young age group, especially among those with overweight or obese BMI (by BMI for age standards). May et al examined the NHANES data from 199-2008, and discovered that among 3383 adolescents (12-19 years old) included, 22% had borderline high (110-129 mg/dL) or high (≥130 mg/dL) LDL cholesterol (38). Additionally, 6% of this sample had low (<35 mg/dL) HDL cholesterol. Another study of 3125 NHANES participants, this from 1999-2006, found at least one abnormal lipid level in 20.3% in these 12-19 year olds (7). Separating by BMI for age category, 14.2% of the normal BMI group, 22.3% of the overweight group, and 42.9% of the obese group had at least one abnormal lipid level.

A good deal of research connects adiposity measures with abnormal blood lipid values in adolescent populations. For example, a study of 948 Saudis aged 10-17 years found significant positive correlations between BMI and TG, TC, LDL cholesterol, and a significant negative correlation between BMI and HDL cholesterol (39). A study of Brazilian children found similar results when examining 113 girls ages 14-19 years. Pereira et al found a significant positive correlation between BMI and LDL cholesterol, along with significant positive correlations between WC and LDL cholesterol, TG, and a significant negative correlation between WC and HDL cholesterol (40). Another study

from Nellore, India recruited 62 obese (by BMI for age standards) adolescents and 24 aged-matched normal weight controls, finding significant differences among the two groups (41). TC, LDL cholesterol, and TG were all higher in the obese group, while HDL cholesterol was lower in the obese group. A recent small study from Lehigh Valley Hospital in Pennsylvania found very similar results after comparing 61 obese (by BMI for age standards) non-diabetic 12-18 year olds and 25 normal weight, aged-matched controls (31). The obese group had significantly higher SBP, CRP, TC, LDL cholesterol, and TG, with significantly lower HDL cholesterol. An Arizona based study of 123 nondiabetic Latinos (mean age 16.3 years) participating in the Arizona Insulin Resistance Registry found those with normal BMI for age had significantly lower TG and oxidized LDL cholesterol, as well as higher HDL cholesterol (42). Moreover, in multiple linear regression analysis, WC was a significant predictor of oxidized LDL cholesterol. Posadas-Sanchez et al examined a larger group of Mexican public school students (n=1846), aged 12-16 years, finding similar results (43). The researchers found that obese (by BMI for age standards) adolescents had the highest prevalence of four different abnormal blood lipid value cutoffs- LDL cholesterol ≥130 mg/dL, TG ≥150 mg/dL, TC ≥200 mg/dL, and HDL cholesterol ≤35 mg/dL. In addition, the researchers found WC was negatively associated with HDL cholesterol, but positively associated with LDL cholesterol and TG in multiple regression analysis. In Portugal, Teixeira et al studied 159 adolescents, 9-18 years old, and found significant positive relationships between BMI and LDL cholesterol, TG, as well as significant negative relationships between both BMI and WC with HDL cholesterol (44). Another study of 167 youths from the greater

Pittsburgh area identified significant positive relationships between both BMI percentile for age and WC with SBP, DBP, TG, and significant negative relationships with HDL cholesterol (45). Additionally, risks for abnormal blood lipids and HTN were examined across 1717 eighth grade students from 12 predominantly minority schools in TX, CA, and NC (46). The researchers found as BMI increased from normal (by BMI for age standards) to overweight to obese, risk for Pre-HTN, HTN, low HDL cholesterol (≤35 mg/dL), and high TG (≥150 mg/dL) significantly increased. In a sample of contrasting demographics, Plourde found BMI was significantly positively related to TC, LCL cholesterol, TG, and significantly negatively related to HDL cholesterol in a case-control study of 1009 Caucasians aged 9-19 years, in hospitals associated with Montreal and McGill universities (47).

Findings from examining large, multifactorial research projects show agreement in results regarding the connection between adiposity and blood lipids in adolescence. For example, Keefer et al found that among a sample of 727 adolescents, 15-17 years of age, from NHANES 2003-2004, BMI was significantly positively related to TC and SBP (32). Spinneker et al found BMI was significantly positively related to LDL cholesterol, TG and significantly negatively related to HDL cholesterol among 1076 subjects, ages 12.5-17.49 years, from the HELENA study of ten centers across Europe (48). In the National Heart, Lung, and Blood Institute (NHLBI) Growth and Health study, 2379 girls ages 9-18 years old were examined (49). Those in the obese (by BMI for age standards) group had significantly increased risk for both systolic and diastolic HTN (SBP, DBP $\geq 95^{th}$ percentile for age), HDL ≤50 mg/dL, and TG ≥130 mg/dL. Results from 547

adolescents 13-18.5 years old from the AVENA study of five centers in Spain found in both boys and girls BMI was significantly positively associated with LDL cholesterol and significantly negatively related to HDL cholesterol, while WC was significantly positively related to TG and significantly negatively associated with HDL (50). More specific to gender, BMI was significantly positively related to TG in boys only, while WC was significantly positively related to LDL cholesterol in girls only. Examining 13-16 year olds from the Kiel Obesity Prevention study in Germany, researchers related BMI and WC to BP (n=3174) and blood lipids (n=536) (51). BMI and WC were significantly positively related to SBP, DBP, TG, and significantly negatively related to HDL cholesterol. In Australia, researchers examined 1139 14-year olds from Perth, and found that in both boys and girls, those with elevated BMI (by BMI for age standards) or WC (by WC for age standards) were significantly more likely to have elevated LDL cholesterol and TG, and below optimal HDL cholesterol (52). Moreover, those with an overweight or obese BMI and elevated WC were more significantly more likely to have elevated LDL cholesterol, TG, SBP, and below optimal HDL cholesterol. Lastly, results from Project Heartbeat! based out of The Woodlands and Conroe, TX show significant relationships between BMI and WC with blood lipids (53).Among the sample of 8-18 year olds (n=678), multiple regression analyses identified significant positive relationships between WC and BMI with TC, LDL cholesterol, TG, and significant negative relationships between WC and BMI with HDL cholesterol. Both BMI and WC appear to be useful predictors of risk factors for cardiovascular disease.

Blood Pressure and Cardiovascular Disease Risk in Adolescents

Another strong independent risk factor for cardiovascular disease is persistently elevated blood pressure. Hypertension, or elevated blood pressure, is highly related to atherosclerosis (8,54–57). Consistently elevated blood pressure leads to weakening and narrowing of blood vessels, potentially allowing more plaque buildup and blockage. Specifically, Berenson et al. (also noted above) found correlations to plaque presence were strong with systolic blood pressure (r=0.55), but weak with diastolic blood pressure (r=0.22) (8). Hypertension is known to lead to stroke, myocardial infarction, and cardiac failure in the adult population (58). While these are not major occurrences among youths, hypertension in adolescence is highly predictive of hypertension in adulthood (59,60).

Rates of hypertension and prehypertension among the adolescent population have not progressed to match the rates of overweight and obesity, but are still of consequence. Among 362 adolescents, aged 12-19 years, data from the NHANES 2007-2008 showed a rate of prehypertension of 10%, and hypertension of 3% (38). Data from NHANES 2003-2006 among 2619 13-17 year olds showed 13.6% and 2.6% prevalence of prehypertension and hypertension, respectively (61). Additionally, 3.2% of 6790 Houston school students, 11-17 years of age, were hypertensive in a study following the students from 2003-2005 (62). Also, 15.7% of those students were pre-hypertensive. Another study of 1020 students found a prevalence of 11.5% and 2.5% of prehypertension and hypertension, respectively (63). An additional study of 5102 schoolchildren found a somewhat higher rate of hypertension in 10-19 year olds, at 4.5% (64). While the US

hypertension prevalence among adolescents was consistently between 2.5% and 4.5%, international data was more variable. Among international research on adolescents, hypertension rates ranged from 2.53% (65), to 3.1% (66) to 5-6% (67) to 8.12% (68) across Hungary, China, Canada, and Brazil. Lastly, a much higher rate of hypertension was found in a study from Lisbon, Portugal at 34%, with an additional 12% prevalence of prehypertension (69). However, the authors noted that the population of Portugal may have a significantly higher prevalence of hypertension in the adult population (nearly 40%) than surrounding nations.

Relationships Between Dietary Intake and Cardiovascular Disease Risk in Adults

While associations between CVD and multiple dietary components, including specific fats, total fat, total energy, fruits and vegetables, sugar-sweetened beverages, whole grains, and multiple micronutrients, have been established, no single class of foods or specific nutrient can be identified as solely responsible for increased CVD risk. Clusters of similar foods and dietary pattern comparisons have been utilized to attempt to isolate whole diet trends, such as low sugar, high fat, convenience, Mediterranean, and Western diets. However, results from studies using these clusters have been somewhat inconsistent, and these clusters often fail to account for dietary adequacy of necessary vitamins and minerals. As such, aggregate scores of whole diet quality, like the Healthy Eating Index, have been developed to encompass the full complement of recommend dietary intakes for optimal health. (See page 17 for a detailed description of the HEI.)

The American Heart Association (AHA) promotes a number of dietary principles to reduce CVD risk, and has identified many specific diet components that may have cardio-protective or pathogenic influences (70). Firstly, the AHA recommends limiting total calorie intake to achieve or maintain a healthful body weight. Next, consumption of fruits and vegetables, in high quantity and a variety of choices, as well as whole grain and high fiber foods is recommended. Certain types of fats and proteins are promoted, particularly fish and plant-based ones, while others, particularly fats from dairy, meat, and poultry, are discouraged. Fish is recommended at least twice per week. Trans fats from processed oils (like margarine) and saturated fats are encouraged to eat in very limited quantities, and be replaced with unsaturated plant fats, like olive oil. Limiting both sugar and salt added to foods are recommended. Lastly, moderation when choosing alcohol is highly recommended. The AHA does additionally note the potential benefit of fish oil supplements and plant stanols/sterols for those with dyslipidemia. Most of these dietary components noted above apply generally to improving the CVD risks that will be examined in this study.

Elevated BMI and WC are both predominantly functions of excess adiposity from energy imbalance. As such, it stands to reason that the majority of dietary factors associated with BMI and WC include total energy consumed as well as a number of types of foods associated with ability to control energy intake. Positive energy balance is the precipitating factor in weight gain and obesity development, and total energy intake is positively correlated with weight, BMI, and risk for obesity (71,72). Moreover, the role of fat intake in changes in BMI and WC is evident, as saturated fat and total fat are

related to risk for obese BMI (71–75). Energy density of the diet is also positively correlated to waist circumference (76). A number of other dietary components with low energy density and high nutrient density, such as fruits and vegetables, low fat proteins, particularly low fat dairy and fish, and high fiber foods (or fiber itself), like whole grains, are negatively associated with BMI and WC (73,75,77–86). Inversely, foods commonly considered low in nutrient density, including fast foods, fried foods, refined grains, sugar-sweetened beverages, and red and processed meat are positively associated with BMI and WC (79,81,87–90).

Some similar dietary influences exist in relation to blood pressure. A wealth of research on the DASH diet has shown a consistently beneficial pattern for reducing blood pressure, one rich in fruits, vegetables, low fat dairy, high fiber foods, and good sources of potassium, calcium, and magnesium while limited in sodium and animal proteins (91–94). Additionally, the literature consistently supports a positive relationship between sodium intake and blood pressure, as well as a negative relationship between potassium intake and blood pressure across a litany of controlled trials (95–100). Sodium is known to attract water, and therefore increasing sodium concentration in the blood attracts water, increases blood volume, and therefore blood pressure. Potassium is primarily thought to reduce blood pressure by countering the effects of sodium. Slightly less evidence supports the inverse relationships between both calcium and magnesium with blood pressure (97,101–103). In particular, blood pressure reduction with calcium supplementation seems to be weak, and present in only systolic, not diastolic. The blood pressure reduction achieved by magnesium and calcium may be attributable to a

combination of mild natriuresis and diuresis. Lastly, omega-3 fatty acids are also inversely related to blood pressure in a number of studies, with research primarily focusing on fish oil supplementation (104–108).

A variety of food types and patterns have also been linked to blood lipids. In particular, saturated fatty acids, trans fatty acids, and total fat have been historically implicated in affecting blood lipids (25,52,109–111). However, a diet rich in omega-3 fatty acids has been associated with improvements in blood lipids (111). Moreover, emerging research identifies higher carbohydrate diets, particularly those rich in refined carbohydrates and added sugars, as tied to dyslipidemia (52,109,112). By contrast, a dietary profile rich in low fat dairy, fruits, vegetables, nuts and legumes is associated with a more favorable blood lipid profile (52,113,114). In that vein, the HEI is related to a more favorable lipid profile, while the "western" diet is associated with a more deleterious lipid profile (52,115).

Dietary Intake of Adolescents

Based on NHANES 2009-2010, a wealth of recent data is available examining what adolescents are eating (116–118). A few trends emerge within the dietary intake data from NHANES 2009-2010 (116–118). In particular, the adolescent diet is adequate in a few areas, including protein and omega-3 fatty acid intake. However, the adolescent diet appears deficient in a number of necessary minerals and food groups associated with positive health outcomes, while excessive in a number of categories associated with negative health outcomes.

Male calcium intake is near the Recommended Dietary Allowance (RDA) of 1300 mg/day at 1260 mg/day, but female calcium intake is well below the RDA at 948 mg/day. Both male and female adolescents are well below the RDA for magnesium. Male magnesium intake is 299 mg/day and female is 224 mg/day, while the RDA is 410 mg/day for males and 360 mg/day for females. Both male and female adolescents eat an insufficient amount of potassium: 2750 mg/day for males and 2008 mg/day for females compared to an Adequate Intake (AI) of 4700 mg/day. Fiber intake also falls short across both sexes. Male adolescents eat a mean 16.4 g/day of dietary fiber, while the AI is 38 g/day. Female adolescents eat a mean 12.6 g/day of dietary fiber, while the AI is 26 g/day. The low fiber intakes of adolescents may be explained by the low intake of the most fibrous food groups- including fruit, vegetable, whole grain, and legumes. Male and female adolescents intake of these foods was very similar when controlling for calorie intake, with 0.44 cup equivalents (CE)/1000 kcal of fruit for male and 0.45 CE/1000 kcal for female. Vegetable intake was comparable, at 0.47 and 0.56 CE/1000 kcal for male and female adolescents, respectively. Whole grain intake was even lower, at 0.26 CE/1000 kcal for male and 0.28 CE/1000 kcal for female adolescents. Legumes were clearly not a dietary staple, as intake was less than 0.10 CE/1000 kcal for both genders.

Saturated fat intake exceeds the recommended limit of <10% total energy across both genders, as both eat >11% calories from saturated fat. Sodium intake is also well above the AI of 1500 mg/day for both male and female adolescents, at 4211 and 2958 mg/day, respectively. In the case of sodium, male adolescents take in quite a bit more than female, although when sodium is controlled for calorie intake, this difference no

longer exists. Refined grain intake, inversely to whole grain intake, was fairly high at 3.26 and 3.20 CE/1000 kcal for male and female adolescents. Sugar intake was also quite high, accounting for nearly half of all carbohydrate intake for both genders, 161 g/day for male and 117 g/day for female adolescents. This high sugar intake is taken into account within the fairly high empty calorie intake. Empty calories were responsible for 37.46% of total calorie intake for male adolescents, and 38.22% for female adolescents.

The dietary intakes of adolescents revealed in the NHANES 2009-2010 data represent a far from ideal diet pattern to reduce risk for obesity, hypertension, metabolic syndrome, and CVD.

Dietary Intake and Cardiovascular Disease Risk in Adolescents

Although the influence of dietary components on CVD risk in the adolescent is not as well researched as in the adult, a number of factors affecting CVD risk have been examined, with notable results.

Sugar, particularly in beverage form, has been examined by multiple researchers for its impact on adolescent weight, adiposity, and CVD risk. Links have been established between increasing sugary beverage or fructose intake and increasing BP, BMI, WC, and body weight in adolescents. A study examining 5033 boys and 4400 girls aged 10-19 years used food frequency questionnaires to assess sugar-sweetened carbonated beverage intake. WC and BMI significantly positively correlated with these beverages in boys only (87). Additionally, a longitudinal study of 1210 black and 1161 white girls measured soda consumption at annual visits from age 9 or 10 until age 19 years using three day

food diaries. Over time, increases in soda consumption from baseline to endpoint predicted the highest increases in BMI during the same timeframe (119). In Germany, research of 249 9-18 year olds, using weighed food records, found an increase in energetic beverage consumption was associated with an increase in BMI standard deviation score (120). A Jamaican study of 1317 15-19 year olds used questionnaires about dietary habits administered in the home to examine dietary data and anthropometrics. Sweetened beverage consumption greater than 1 per day was associated with increased odds of overweight by BMI for age (121). Fiorito et al. performed a longitudinal study with 170 non-Hispanic white girls, assessing dietary intake using three 24 hour diet recalls biennially from 5 to 15 years old. The researchers found sweetened beverage intake at age 5 was positively associated with WC, weight, and % body fat (122). Gillis and Bar-Or studied 91 obese and 90 non-obese 4-16 year old Caucasians. Diet history interviews conducted with a Registered Dietitian revealed that obese participants consumed significantly more sugar-sweetened drinks (123).

Study of NHANES 1999-2004 data on 4867 12-18 year olds revealed increases in systolic BP z-scores moving from the lowest to highest sugar-sweetened beverage consumption (124). A case-control study of 174 obese ($\geq 97^{th}$ percentile BMI for age) and 174 normal weight Spanish adolescents (mean age 11.6 years) found consumption of >4 servings sugar-sweetened carbonated beverages was significantly associated with the presence of obesity (125). Moreover, the researchers found each additional serving of such beverages increased relative risk for obesity by 69%. Lastly, a study of 559 14-18 year olds in Augusta, GA used 4-7 24 hour diet recalls to assess fructose consumption.

The researchers found significant positive associations with visceral adipose tissue (measured by MRI), and significant positive upward trends across tertiles of fructose intake with systolic BP and CRP (126). Lastly, an examination of 12-19 year olds from NHANES 1999-2004 (n=2157) found added sugars were positively related to LDL cholesterol and TG (112). Additionally, in this sample, added sugars were negatively correlated with HDL cholesterol.

A number of studies also examined the relationships between dietary intakes of certain food groups, particularly fruit, vegetable, dairy, and grain. Negative associations have been found between all these groups and BP, CRP, BMI, and WC. The same Jamaican study above found fruit intake of less than one serving per day was significantly associated with high waist circumference in those adolescents (121). Additionally, a study of 21,111 Iranian students aged 6-18 years found significant inverse associations between food groups and BMI based on self-administered food questionnaires. The researchers found plant proteins and vegetables were negatively related to BMI in boys, and dairy and fruit were negatively related to BMI in girls (127). Findings from the HELENA study of 12.5-17.5 year olds across 10 sites in European countries are in agreement, as dairy intake was negatively associated with WC, and with BMI and TG in females only (114). US research findings are similar, as study of NHANES III data of 1803 adolescents found those who were considered centrally obese ($\geq 85^{th}$ percentile WC for age and sex) had significantly lower dairy, grain, fruit, and vegetable consumption (113). Bradlee et al studied 1500 girls 9-17 years old participating in the NHLBI Growth and Health Study across 3 sets of dietary records, who received follow up of blood lipids

drawn between 18-20 years old (128). The researchers found dairy intake was negatively related to TC and LDL cholesterol, fruit was negatively related to TC, non-HDL cholesterol, and LDL cholesterol, and intake of nuts, seeds, and legumes was negatively related to non-HDL cholesterol and LDL cholesterol. Additionally, combined intake of fruit and non-starchy vegetables was negatively associated with TC, non-HDL cholesterol, LDL cholesterol, and log of TG. Additionally, data from the Framingham Children's Study from 8 years of annual follow up visits beginning from 3-6 years of age revealed more fruit and vegetable intake during the preschool years predicted lower mean systolic BP in early adolescence (129).

Dietary fiber has additionally been researched in the adolescent diet, with negative relationships to adiposity in the trunk region. A cross-sectional study of NHANES 1999-2002 data on 2128 12-19 year olds showed a significant inverse relationship between grams of fiber eaten per 1000 calories eaten and the prevalence of metabolic syndrome. In fact, each quintile increase of fiber intake was associated with a 20% decrease in prevalence of metabolic syndrome in the sample (130). A smaller study of 85 overweight 11-17 year old Latinos used two day diet recalls and magnetic resonance imaging (MRI) to assess dietary fiber intake and visceral adipose tissue. The researchers found a significant inverse relationship between fiber intake and visceral adipose tissue (131).

Saturated fat has been identified in the adolescent research as linked to increased BP and TG. In a study of 1066 Finnish children, beginning from age 7 months to 15

years, BP was measured annually. In the study, 540 children were assigned low saturated fat, low cholesterol diet counseling each year, and 522 were matched controls. The researchers found the intervention group had significantly lower systolic and diastolic BP at age 15 years (132). Additionally, research by Washi and Ageib out of Jeddah, Saudi Arabia showed a significant positive relationship between saturated fat intake and TG (109). In a similar vein, total fat intake was significantly positively related to LDL cholesterol in this sample of 239 adolescents.

Dietary sodium intake in adolescents has been positively associated with BP. A British study of 1658 4-18 year olds measured sodium intake by seven day diet record and compared to BP. A significant positive association was found between sodium intake and systolic BP. Moreover, every 1 gram increase in sodium intake per day was associated with 0.4 mm Hg increase in systolic BP (133). Study of NHANES 2003-2008 data on 6235 8-18 year olds also showed an increase in BP with increases in each quartile of sodium intake. The researchers also revealed a potentially synergistic effect of sodium intake and overweight/obesity on BP and risk for pre-hypertension and hypertension. Among the sample, a 1 gram increase in daily sodium intake led to a 0.097 standard deviation score increase for systolic BP (0.141 increase among overweight/obese). Additionally, odds of pre-hypertension and hypertension increased by 2.0 times between the highest and lowest quartiles of sodium intake (by 3.5 times among overweight/obese) (134). Considering sodium does not directly provide any energy, one study found a more novel relationship between sodium and BMI and WC. Among a sample of 766 white and African American 14-18 year olds, 7 day 24 hour diet recalls were used to determine

sodium intake and dual energy x-ray absorptiometry (DXA) to determine body fat measurements. The researchers found sodium to be significantly positively related to weight, BMI, WC, % body fat, fat mass, and subcutaneous adipose tissue in the sample (135).

Bringing together much of the above, one study clustered eating patterns of 1139 adolescents (all age 14 years) from Perth, Australia into two groups: Healthy vs. Western (52). The Healthy group had a diet lower in total fat, saturated fat, added sugars, and sodium, but higher in protein, carbohydrate, folate, and fiber than the Western group. In girls, the more Western diet was significantly associated with elevated WC, BMI, and total cholesterol. By contrast, in boys, the more Healthy diet was significantly associated with optimal HDL.

Total energy intake plays a key role in weight status, and literature in adolescents identifies correlations between energy intake and BMI, weight, and body fat. Longitudinal study of 12829 US children ages 9-14 years old revealed a trend between energy intake and BMI. From 1996-1999, children returned questionnaires reporting height and weight, and returned food frequency questionnaires. Increases in total calorie consumption from the previous year predicted increases in BMI among the participants (136). Demol et al. randomly assigned 55 obese 12-18 year olds to one of three 1200-1500 calorie diets for 12 weeks. They found a significant decrease in BMI, BMI standard deviation score, and % body fat from baseline to 12 weeks across all diets (137). In India, research examined 96 overweight and obese 12-18 year olds given diets with a 500

calorie per day energy deficit and either 2, 3, or 4 servings of dairy per day for 12 weeks. Regardless of dairy consumption, a significant decrease from baseline to 12 weeks was found in BMI, BMI z-score, weight, % body fat, and total body fat (138). Additional research from Saudi Arabia of 239 adolescents cluster sampled from schools in Jeddah showed relationships between energy intake and blood lipids (109). Kcal intake was positively correlated with TC and TG. Additional, the source of energy was important, as % energy from fat was positively related to BMI for age.

Diet Quality in Adolescents

Although single nutrient analysis or analyses focusing on total calories, fat, protein, and other dietary measures from total diet has provided great use, an effort has been made to incorporate multiple aspects of total diet into a one measure (139–143). Multiple scores/tools have been developed to measure what is known as diet quality, such as the Recommended Food Score, the Diet Quality Index, and the Healthy Eating Index. Diet quality is a single score distilled from multiple components of dietary intake, including various food groups, specific macro and micronutrients. An overall dietary quality score is considered advantageous to more traditional approaches of examining single macro or micronutrients, or energy intake.

The original Healthy Eating Index (HEI) was developed in 1995 by the US Department of Agriculture's (USDA) Center for Nutrition Policy and Promotion (CNPP) and has been widely used in nutrition research (144–147). Since 1995, two updates have been released, the HEI-2010 is the most recent, a twelve-component scoring tool with a

maximum of 100 points possible, developed as a new update for the HEI-2005 (148). Scoring is detailed in Appendix A. Because the HEI-2010 reflects the Dietary Guidelines for Americans as well as emerging research, it is widely considered the most practical and most valid measure of diet quality available (19). As such, this literature review will focus on diet quality studies utilizing the HEI in one of its three iterations: the current HEI-2010, most recent HEI-2005, or original HEI. In the literature below, the iteration used will be noted as HEI-2010, HEI-2005, or HEI (for the original version).

The HEI-2010 offers the benefit of not only measuring adequacy of various types of foods/nutrients, but also the moderation of other foods/nutrients intake. The HEI-2010 provides perspective on adequacy of individual food groups, such as total fruits and dairy, more specific foods within groups, such as greens and beans and seafood and plant proteins, and appropriate distribution of fatty acids (measured as total unsaturated fatty acids/saturated fatty acids). The HEI-2010 also includes a measure of moderation of sodium, refined grain, and empty calories (defined as solid fats, added sugars, and alcohol) consumption (148).

With all versions of the HEI, including the original HEI, the HEI-2005, and the HEI-2010, a score reflecting a good diet is considered 80 out of 100 (143,148,149). This score would reflect near optimal intake of all the food groups noted, as well as limited sodium, limited empty calories, predominantly whole grain intake, and minimal solid fat intake.

Review of diet quality in NHANES data reveals very poor adherence to dietary guidelines, and thus poor diet quality, among the adolescent population. Data from the 2003-2004, 2005-2006, and 2007-2008 NHANES 2-17 year old samples show average diet quality among US children and adolescents to be below 50 in 2003-2004 (n=2996), 2005-2006 (n=3237), and 2007-2008 (n=2703). The mean diet quality scores were 46.9, 47.1, and 49.8, respectively. Total Vegetables, Greens and Beans, Whole Grains, Seafood and Plant Proteins, and Refined Grains showed the worst adherence to recommendations (lowest scores) in each of the three subgroups (2). Similar results were found using the HEI-2005 to analyze NHANES 2003-2004 data of 1623 12-17 year olds (3). In this sample, the average HEI score was 54.8, with worst adherence found in the Sodium, Whole Grains, and Dark Green and Orange Vegetables and Legumes subgroups. Recent research examining data from combined NHANES 2005-2010 data revealed an even lower HEI-2010 score than any of those above. Banfield et al found a mean HEI-2010 score of 43.59 among 14-18 year olds, with worst adherence to recommendations for Fatty Acids, Whole Grains, Refined Grains, and Greens and Beans groups (150).

Another study of diet quality in NHANES data from 1999-2004 found similarly poor adherence to guidelines (151). O'Neil et al found a mean HEI-2005 score of 47.68 in 13-18 year old adolescents (n=4931), and found significantly higher HEI scores with higher Whole Grain consumption. Additional research by O'Neil et al found a significantly higher HEI-2005 score among those who consumed 100% fruit juice than those that did not (4). A sample of 3139 adolescents (age 13-18 years) from NHANES 2003-2006 was examined, finding HEI-2005 scores of 49.6 and 44.4 among 100% fruit

juice consumers (n=1397) and non-consumers (n=1742), respectively. Earlier work by Goodwin et al among 1504 adolescents (aged 11-18 years), from the Continuing Survey of Food Intakes by Individuals 1994-1996, showed original HEI scores of mean 61.8 (147). These findings may reflect improvements in the efficacy of the updated HEI scores, or a negative trend in diet quality among US adolescents. Further study of the Continuing Survey of Food Intakes by Individuals, expanding to a timeframe of 1994-1998 found a mean HEI-2005 score of 48.1 among 1679 10-18 year old adolescents (5).

Additional literature is also available among Brazilian and Turkish adolescent diet quality (152–154). Two Brazilian studies from 2001-2002, both of adolescents in the Sao Paolo area, found an identical mean HEI score of 59.7 (152,153). Acar Tek et al found poorer diet quality among Turkish adolescents (154), with a mean HEI-2005 score of 51.5.

Diet Quality and Cardiovascular Disease Risk in Adolescents

While there is some good data available describing the diet quality of adolescents by HEI, very little research is available to link HEI to chronic disease risk in this population. Among the few that do, only measures of adiposity are correlated to HEI scores, and the populations are not generalizable to other adolescents (146,155–157). In a study of 252 8-18 year old type I diabetics, 3-day diet records were collected from July 2008-February 2009, and analyzed to determine HEI-2005 scores. The mean score was 53.4, and did not correlate directly with BMI for age percentile (155). However, higher fruit and whole grain scores were associated with lower BMI for age percentile.

Research of childhood cancer survivors (n=91; mean age 18.7 years) and their sibling controls (n=30; mean age 20.7 years) showed HEI-2005 scores of 55.5 and 53.3, respectively (156). The subjects were recruited from the Finger Lakes region between 1999-2003, with HEI-2005 score determined from 3-day diet record, and HEI-2005 score was compared across BMI groups and with body fat percent determined by DXA. The researchers found a significant difference in HEI-2005 scores between the overweight and obese groups, with the obese group having significantly lower HEI-2005 scores. Additionally, HEI score had a negative correlation with body fat percent (β= -0.19).

A similar relationship between body fat and HEI score in a study of two samples of low-income, African American adolescents (146). The HEI was determined from the Youth/Adolescent FFQ administered in both the Challenge Study (n=196; 11-16 years old) from April 2001-May 2004, and the Three Generation Project (n=121; 14-19 years old) from June 1997-September 1999. Mean HEI scores were 62.83 and 59.93 among the Challenge Study and Three Generation Project subjects, respectively. In the Challenge Study only, body fat percent and abdominal fat percent were measured via DXA, and both were negatively correlated with HEI (r= -0.17 for body fat percent; r= -0.19 for abdominal fat percent).

One study has identified a significant relationship between HEI and blood lipids in the adolescent population. Based in Iran, the Tehran Lipid and Glucose Study from 2008-2011 examined 706 adolescents aged 10-19 years (115). The researchers found that HEI-2005 score was significantly negatively related to TG and positively related to HDL

cholesterol. While the research analyzing the relationship between adolescent HEI and CVD risk is limited, evidence is emerging to link adiposity measures to HEI in this population.

Race, Gender, and SES

Research reveals distinct racial differences among adolescents in CVD risks. In particular, African Americans tend to have higher blood pressure and BMI compared to white Americans (158–160). Additionally, African American girls have higher prevalence of overweight (BMI> 85th percentile for age) and severe obesity (BMI>35 kg/m^2 or 120% of 95th percentile BMI for age) in ages 12-19 years compared to other racial groups (49,161). Other racial differences in CVD risk, seen in adulthood, are not necessarily identified within the adolescent populations.

Differences in diet quality are noted across socioeconomic status (SES) as well as gender. While this pattern does not necessarily hold true elsewhere, in the US and Canada, boys tend to have poorer diet quality than girls (162–165). Additionally, increasing SES is consistently associated with increasing diet quality (163,166–168).

Gaps in Literature

Minimal research is available linking diet quality in the adolescent population to any chronic disease risk markers. Moreover, no data is currently available directly addressing the relationship of diet quality (i.e., HEI) and many CVD risks in this age group, such as BP and blood lipids. Although some evidence has recently emerged

linking these variables in adult samples, research examining the potential role of diet quality on CVD risks in adolescents is in its infancy, and has not been thoroughly studied as a predictor of CVD risk in general.

While a relationship has been established between adiposity and diet quality, research examining the interaction of these two variables to predict chronic disease risk is still unavailable. Considering the independent roles both diet quality and obesity play in origination and progression of multiple chronic diseases, particularly CVD, this interaction could be very informative. Finally, research currently available linking diet quality and adiposity is also incomplete. The samples utilized in previous studies are very unique (i.e., Type I diabetics, cancer survivors and low-income African Americans) and not representative of the adolescent population as a whole.

Purpose

The long-term goals of this project were to elucidate the influence of diet quality on the risk of cardiovascular disease in adolescents. This research examined differences in diet quality among adolescents displaying CVD risks and those not, as well as the relationships between diet quality and a number of CVD risks. Furthermore, this research explored potential interaction effects between diet quality and adiposity in predicting other CVD risks.

Limitations

The study has a few distinct limitations. In particular, the data is measured at a single time point, not allowing temporal precedence to be established. Additionally, the research is non-experimental so no cause and effect relationship can be concluded. Also, the sample is almost exclusively two races, Caucasian and African American, which is not representative of the US, nor North Carolina. Information to determine Tanner stage of physical development is not available, leaving us to assume all the 16 year olds in the sample are at the same stage of development. Measures for some variables may not be ideal or directly comparable to other literature as well. In particular, Hollingshead score is used to assess socioeconomic status, which is not a direct reflection of income, the most commonly utilized assessment in literature.

Also of concern is the lack of physical activity data, which could play a key role in the progression or prevention of CVD risk. Moreover, the CVD risk variables measured are not direct measures of atherosclerosis or other detrimental changes to the cardiovascular system. As such, CVD is inferred as more likely when possessing these risks, rather than having true knowledge of CVD presence.

Finally, obtaining dietary recalls from adolescents brings with it a host of potential complications, whether simply not answering the collectors' calls or not being willing to provide accurate data. Due to the increased incidence of disordered eating behaviors among adolescents compared to adults or younger children, accuracy of information provided may be of concern.

CHAPTER III

DIET QUALITY AND ADIPOSTITY IN ADOLESCENTS

Introduction/Background

As obesity rates in the United States peak among children and adolescents, diet quality continues to be poor. While 18.4% of U.S. adolescents, aged 12-19 years, are now considered obese (1), diet quality measurements consistently reveal well below optimal scores (2–5). Additionally, longitudinal research indicates dietary patterns from adolescence may persist into adulthood (6).

The prevalence of obesity among adolescents, while not as high as adults, is still a public health concern. Ogden et al reported 33.6% of US adolescents are overweight, with 18.4% obese (1). These results are based on data from the National Health and Nutrition Examination Survey (NHANES), covering 2009-2010, with overweight and obesity based on body mass index (BMI) for age ($\geq 85^{th}$ percentile overweight; $\geq 95^{th}$ percentile obese).

A recent review examined abdominal obesity specifically across many nations, using waist circumference (WC), as the indicator (28). The cutoff point for abdominal obesity diagnosis using WC had no consensus in this review, and 14 different cutoff points were utilized across 1977-2007, with the most common cutoffs being $\geq 90^{th}$ percentile of the sample (as proposed by Cook et al (29)), $\geq 95^{th}$percentile of the sample,

$\geq 90^{th}$ percentile of the country, or $\geq 70^{th}$ percentile of the country. This review found a wide range in obesity rates, from 3.8% to 51.7% among 10-19 year olds. Among developed nations, a narrower range was reported, 8.7% to 33.2%.

Positive energy balance is the precipitating factor in weight gain and obesity development. Among adults, total energy intake is positively correlated with weight, BMI, and risk for obesity (71,72). Moreover, the role of fat intake in changes in BMI and WC is evident, as saturated fat and total fat are related to risk for obesity in adults (71–75). Energy density of the diet is also positively correlated to waist circumference among adults (76). A number of other dietary components with low energy density and high nutrient density, such as fruits and vegetables, low fat proteins, particularly low fat dairy and fish, and high fiber foods (or fiber itself), like whole grains, are negatively associated with BMI and WC (73,75,77–86). Inversely, foods commonly considered low in nutrient density, including fast foods, fried foods, refined grains, sugar-sweetened beverages, and red and processed meat are positively associated with BMI and WC (79,81,88–90). However, substantially fewer studies have examined these same relationships in adolescents. Of those that do, sweetened beverages are implicated in increased adiposity, as well as higher calorie intake, proportion of energy intake from fat, fast foods, and sodium (87,109,119–123,135–138). By contrast, some research in adolescents suggests that increased fruit, vegetable, dairy, and whole grain consumption are associated with lower adiposity (113,114,121,127).

Another way to examine the relationship of diet with adiposity is to use a validated overall diet quality measure. The Healthy Eating Index-2010 (HEI-2010) is a validated measure of total diet quality, based on the current Dietary Guidelines for Americans (19). Review of diet quality in NHANES data reveals very poor adherence to dietary guidelines, and thus poor diet quality, among children and adolescents (2). Data from the 2003-2004, 2005-2006, and 2007-2008 NHANES 2-17 year old samples show average diet quality, using HEI-2010, among US children and adolescents to be below 50 out of a possible 100 in 2003-2004 (n=2996), 2005-2006 (n=3237), and 2007-2008 (n=2703). The mean diet quality scores were 46.9, 47.1, and 49.8, respectively, with a score of 80 or higher indicating a "good" diet. Banfield et al found a mean HEI-2010 score of 43.59 among 14-18 year olds in NHANES 2005-2010 data.

While there are recent reports of diet intake and quality among adolescents, there is a paucity of research exploring the associations between diet and adiposity in adolescents. Therefore, considering the poor quality of diet and high obesity rate in the adolescent population, the purpose of this study was to examine the relationships among diet and adiposity in a diverse sample of 16 year olds. The specific aims were to identify differences in diet quality of obese and non-obese adolescents, determine the relationships among diet quality with body mass index and waist circumference, and identify relationships between specific nutrients or food groups with adiposity, controlling for race, gender and socioeconomic status.

Materials and Methods

Sample

A subsample of adolescents participating in "The Right Track" research study was used for this dissertation research study. "The RIGHT Track" study examines the role of childhood self-regulation in the context of family processes, in the development of adolescent psychopathology (169).

Since the onset of "The RIGHT Track" study in 1997, over 450 families have been followed. Currently, the participants range in age from 15-19 years. Data collection has previously occurred at ages 2, 4, 5, 7, and 10 years, with further data collection to occur at ages 15 and 17 years for "The RIGHT Track."

The sample was originally recruited from the greater Greensboro, NC community, including daycare centers, county health departments, WICs, etc. in 1997 for children aged 2 years for cohorts one and two (169). Cohort three was recruited in 1998 at 6 months age. All cohorts were oversampled to include more children identified by the caregiver with externalizing behavior problems. Externalizing behavior problems include fighting, cursing, stealing, destruction of property, refusal to follow either formal or informal rules, running away from home, and generally impulsive behaviors. The samples were also recruited with an effort to maintain approximately equivalent numbers of participants of each sex. Children with diagnosed developmental abnormalities or chronic diseases were excluded from the sample. It is a convenience sample, approximately 70% Caucasian, and the remainder predominantly African-American.

In terms of socio-economic status, the sample is highly diverse. The Hollingshead social status scores (170), a tool to estimate socio-economic status based on the parent's education level and current employment, range among the sample from 9-66, with a mean of 43.5. The score is obtained by multiplying a possible score of 1-7 for educational attainment (1 being below high school and 7 being a graduate degree) by 3, then multiplying a profession score of 1-9 (1 being unskilled labor and 9 being CEO, professor, or commissioned officer in the military) by 5, and finally summing the two. Given that the minimum score is 8 and the maximum score is 66, the sample represents both the highest socio-economic status and very near the lowest. Moreover, of the fathers of children in the sample, 25% did not complete high school, while 32% attended some college.

The proposed study focused on cohorts 2 and 3 of "The RIGHT Track" sample, which, at first contact (age 2), contained 293 participants. By age 10, the sample had reduced to 257 participants (88% retention rate). Two hundred (not sure of the exact number yet) participants completed most of the measurements at age 16. Of these, 163 completed dietary recalls.

The data obtained for this research occurred entirely within the "RIGHT Track" participants' 16^{th} year of age, from February 2014 to October 2015.

Human Subjects Protections

This study has been approved by the Institutional Review Board of the University of North Carolina at Greensboro. All adolescent participants for this study provided

assent to participate. All parents/guardians of the minor participants provided consent for the adolescent to participate.

Demographics and Anthropometrics

During each adolescent participant's 16th year of age, he/she visited the UNCG Exercise Physiology laboratory with a parent/guardian present. The adolescent participant was fasted for at least 10 hours of food, but not water. As such, these visits were almost exclusively scheduled in the AM hours between 8 am to 12 pm to ease participant discomfort.

After first arriving at the lab, informed consent was obtained from the parent, and informed assent was obtained from the adolescent participant. Next, the parent and adolescent signed forms accepting their compensation for participation, in the form of a Target gift card for the adolescent and check for the parent ($50 each for the parent and adolescent).

A research assistant of the same gender as the participant measured the participant's height, weight, and waist circumference. Height was measured with a Seca 222 stadiometer to the nearest 0.5 cm, with the participant shoeless. Weight was measured via Seca 770 electronic scale to the nearest 0.01 kg. The participant wore neither shoes nor jackets, with pockets emptied. Waist circumference was measured with Baseline 12-201 Gulick Measuring Tape directly against the skin, at the natural waist, the narrowest point of the abdomen between the lower rib cage and iliac crest.

Diet Recalls

Prior to exiting the lab, a research assistant instructed the adolescent participant on the process of the diet recalls, which included calls from the University of Chapel Hill Nutrition Obesity Research Center staff. The research assistant also provided the adolescent participant with a booklet of common guides for portion sizes (which was utilized during the diet recall interview,). Lastly, the research assistant ascertained the best contact telephone number and best timeframes to contact the adolescent participant. We anticipated this to be predominantly cell phones. As such, the adolescents were encouraged to keep the food amounts booklet in a backpack, book bag, or large purse that was near them most of the day where possible.

Participants were called by trained and certified staff of the UNC-CH Nutrition Obesity Research Center to collect diet recalls on two weekdays and a weekend day. These recalls were completed within two weeks of completion of the laboratory visit described above. The participants had been instructed to keep the portion booklet provided near a phone or on their person during the timeframes they preferred to be contacted. This booklet provided two-dimensional pictures of many common foods, food measurements, food containers, and food shapes, and participants could use it as a guide to aid in accuracy of the measurement estimation of foods eaten and beverages drunk. If the participant was without the portion booklet at the time of the call, the staff continued to collect the dietary data, prompting with references for portion sizes where appropriate.

The diet recalls collected utilized the University of Minnesota's Nutrition Data System for Research (NDSR), which followed a standard script and multiple pass method. This method allowed multiple opportunities to recall food, beverage, and dietary supplement intake over the previous day's 24-hour period studied. All recall data was overseen by the Nutrition Obesity Research Center's program coordinator for quality assurance.

As we anticipated diet recall data to be difficult to obtain, and follow through with all three diet recalls especially difficult, the incentive system was tiered for completion. The adolescent participant received Target gift cards equaling $10 for completing the first diet recall, $15 for the second, and $20 for the third, with a max total of $45.

Diet Quality

Diet quality was assessed with the HEI-2010, a multi-component scoring tool to assess an individual's diet quality based on data provided via diet recall, food record, or food frequency questionnaire (FFQ). The original HEI was developed in 1995 by the US Department of Agriculture's (USDA) Center for Nutrition Policy and Promotion (CNPP), and since has been updated twice. The HEI-2010 is the most recent update, and is considered the most valid measure of whole diet quality available (19).

The HEI-2010 contains twelve-components, for a maximum score of 100 points. Each component can receive a score ranging from 0 for a minimum up to 20 points maximum (depending on assigned maximum point total). Scores between the minimum and maximum values are prorated based on intake. Individual component scores are

summed for a total score of minimum 0 up to 100 points maximum. Scores above 80 are considered representative of a "good diet," while scores of 50 or below are considered "poor," and those between 50 and 80 are considered "needing improvement" (148). Nine components represent the adequacy of certain food groups, while three represent the moderation of intake of certain foods associated with negative health consequences. Eight of the nine adequacy scores are based on 1,000 kcal intake. Basing scores off kcal density limits the bias toward diets with more total food, and therefore, greater likelihood of the select food group intake. The Fatty Acids group is scored as a ratio comparing unsaturated and saturated fat. More specifically, it is expressed as the sum of grams of polyunsaturated fatty acids and monounsaturated fatty acids, divided by grams of saturated fatty acids. Among the moderation components, refined grains are any non-whole grains. Sodium is scored based on eating under a maximum intake of 2000 mg per 1,000 kcals. Lastly, the empty calories component reflects a percentage of kcal intake from solid fats, alcohol, and added sugars. However, alcohol is not used in this component unless intake is greater than 13 grams per 1,000 kcal. A lower percentage of kcal from empty calories improves the score of this component up to the maximum 20 points.

The researcher utilized outputs from the NDSR to calculate the HEI-2010 score, based on average intake of foods and nutrients across all days' dietary information obtained. A detailed outline of the conversion process is provided in the Appendix A. Generally, each average intake of a food group was converted to cup or ounce equivalents, as appropriate, then divided by average energy intake and multiplied by 1000

to standardize to an intake per 1000 kcals. HEI-2010 score was produced based on the percentage consumed of the minimal intake for a full score. A similar process was utilized to determine sodium, refined grain, and empty calories scores, although scoring was based on the percentage consumed of the maximal intake for a full score.

Statistics

SPSS Statistics (Microsoft, version 20) was used to perform all statistical analyses. Obese was defined as BMI $\geq 95^{th}$ percentile for age, and abdominally obese as WC $\geq 90^{th}$ percentile for age based on NHANES data. Participants with measurements below these cut-offs are considered non-obese. It should be noted that this WC for age cutoff at age 16 are similar to, but higher than the WHO WC cut off used in adults to categorize risk for chronic disease (approx. 106 cm for males and 102 cm for females compared to 100 cm and 90 cm). Inversely, this BMI for age cutoff at age 16 reflects a similar, but lower cut off compared to the WHO standard (approx. 27.5 kg/m^2 for male and 29 kg/m^2 for female compared to 30 kg/m^2 for both). ANOVA was used to determine differences in gender, race, SES, HEI-2010 scores, and nutrients between obese and non-obese groups.

Correlations were examined among all variables, including adiposity, specific nutrients, food groups, and HEI-2010 scores, to identify multicollinearity, and significant correlations between the variables. Regression analyses to identify models for diet and adiposity were run, with race, gender, and SES considered as covariates. We ran stepwise regression analyses of BMI and WC as dependent variable with HEI-2010 scores, total

energy, total fat, saturated fat, omega-3 fatty acid, refined grain, whole grain, fruit, vegetable, fiber, sweetened beverage, and empty calorie intakes as independent variables. Sweetened beverages included beverages with added sugar such as soda, lemonade, kool aid, sweet tea, sports drinks, and sweetened coffee drinks, but excluded fruit juice and flavored milk. Results were considered significant at the $p<0.05$ level.

Results

A total of 163 adolescents agreed to participate in diet recalls. One hundred forty nine participants provided three days of recall, six provided two days of recalls and eight provided only one day of recalls. There were no significant differences in the average HEI scores nor any other dietary variables analyzed between these three groups. One hundred forty three participants provided anthropometric data by coming to the UNCG Exercise Physiology Lab for measurements. An additional 19 self-reported height and weight data either due to excessive distance to travel to the lab or scheduling conflicts. It should be noted that correlations between self-reported BMI (from the provided height and weight) and BMI determined from height and weight measurements taken at the lab were very strong ($R=0.961$, $p<0.001$).

Table 1 highlights sample characteristics by BMI and WC status. The final sample totaled 98 females, 65 males, 107 white participants, and 56 non-white participants. Four individuals were considered underweight by BMI for age. A significantly ($p<0.05$) higher proportion of nonwhite participants were obese by both BMI and WC than white participants. Mean SES for the sample was 45.4±13.1 (range of

0.38-66) with significant differences observed in SES between obese by BMI for age and non-obese, obese by WC for age and non-obese participants, as well as by race. SES differences between obese by BMI and obese by WC compared to non-obese participants are also listed in Table 1. Racial differences in SES were identified, as non-white participants had significantly lower (40.5±13.8) SES than white participants (47.6±12.3) at $p<0.01$.

HEI-2010 score differences in obese participants by BMI for age and non-obese are reported in Table 2. No significant differences in HEI-2010 total scores or component scores were observed between obese by BMI for age and non-obese, between obese by WC for age and non-obese, or between genders. The mean HEI-2010 score for the entire sample was 49.2±12.0, with only two of the 163 participants meeting the recommended diet quality of 80 or higher. Mean HEI-2010 score for males was 47.8±10.9, while mean HEI-2010 for females was 50.1±12.7 ($p=0.222$).

Racial differences in consumption in diet quality also emerged in this sample. White participants had a higher HEI-2010 score (50.8±12.6 vs. 46.0±10.1) compared to non-white participants at $p<0.01$. Multiple component scores were different across race as well, with whole fruit (1.8±1.9 vs. 0.9±1.4), greens and beans (1.8±2.1 vs. 1.2±1.8), whole grains (4.0±3.0 vs. 2.9±2.9, at $p<0.01$), dairy (6.9±3.1 vs. 5.6±2.8), and seafood and plant protein (1.9±2.1 vs. 1.3±1.8) scores all significantly higher in white participants compared to nonwhite participants at $p<0.05$. Inversely, white participants had a significantly lower than nonwhite participants at $p<0.05$. Lastly, white participants had

significantly higher intake of dietary fiber (14.8±7.3 vs. 11.7±5.1) compared to nonwhite participants at $p<0.05$.

Table 3 shows a variety of nutrient and food group intakes by obesity status. Of note, only sweetened beverage intake was significantly different comparing obese by BMI for age and non-obese, with the obese group drinking more sweetened beverages (note this variable excluded fruit juice and flavored milk) and eating less fruit. No significant differences were observed between the obese by WC for age group and the non-obese group.

Table 4 displays the results of stepwise linear regression analysis controlling for race, gender, and SES. Total fruit intake emerged as the only significant dietary predictor of BMI, while race, but not SES or gender, was also a significant predictor of BMI in the model. Race, gender, and total protein intake, but not SES, emerged as significant predictors for WC.

Table 5 identifies significant correlations after partial correlations analysis controlling for race, gender, and SES. BMI and WC are significantly positively correlated. BMI is significantly negatively correlated with total fruit and dietary fiber. WC is significantly negatively correlated with total fruit and total protein.

Discussion

Among this diverse sample of 16 year olds, obese adolescents drank more sweetened beverages than non-obese adolescents. Additionally, when controlling for

race, gender and SES, there was a significant negative relationship between fruit, and fiber intakes and BMI. Only fruit and protein intake were significantly, negatively associated with WC.

Diet quality of the sample was similar, but slightly higher than that presented in other US research, where mean HEI-2010 scores range from 43.59 in a sample of 14-18 year olds to 49.8 in 2-17 year olds (2–4,150,163). The differences in diet quality by obesity status revealed in this research agree with two previous studies. Landy et al identified lower HEI-2005 scores among obese late adolescents compared to overweight in a sample of 121, and a negative relationship between HEI-2005 score and body fat percent determined by dual energy X-ray absorptiometry (DXA) (156). Hurley et al found negative relationships between HEI scores of a sample of 196 11-16 year olds and both body fat percent and abdominal fat percent determined by DXA (146).

Moreover, this sample had similarly poor adherence to multiple components of the HEI-2010 compared to previous research. In particular, worst adherence in this sample is in the total fruit, whole fruit, greens and beans, whole grains, seafood and plant proteins, sodium, and refined grains groups. Although previous research shows better adherence to total and whole fruit recommendations and worse to total vegetable recommendations than expressed in this sample, multiple studies show very poor adherence to greens and beans, whole grains, seafood and plant proteins, refined grains, sodium, and sodium components among adolescents (3,150,163).

Findings from this research highlighting the greater intake of sweetened beverages among obese adolescents and negative relationship between fruit intake and BMI tend to be in agreement with previous research in both adults and adolescents. Sweetened beverage intake has a consistently positive relationship with adiposity established in the literature in both adults (79,81,87–90) and adolescents (87,119–123). Fruit intake has been identified as protective against adiposity in multiple studies, though predominantly in adults (82–86), rather than adolescents (113,121). Additionally, increasing fiber intake has been linked to decreasing adiposity in prior research (73,77,78). The inverse relationship of protein and WC is identified in some available research, as Kelishadi et al noted similar findings in a sample of Iranian adolescents. However, the adult literature reveals conflicting results of the relationship between protein intake and WC (16,171–175). Work by Ambrosini et al also agrees with these findings, as adolescents in that sample who consumed a diet richer in fruit and protein while lower in sugar had lower BMI and WC (52).

Although findings here agree with previous literature regarding sweetened beverage and fruit intake, this research did not identify relationships between adiposity and a number of other dietary components found in prior studies. In particular, multiple studies in adults find significant positive relationships between BMI and/or WC with calorie intake (71,72), total, and saturated fats (71–75). This may be an issue of under-reporting of energy intake among the obese participants in this study, and significantly greater under-reporting has been observed in the obese in previous research (176–179).

Other research found inverse relationships between BMI and/or WC with intake of vegetables (82,83,85,86) and whole grains (80,81).

This study highlighted differences across racial groups and SES in obesity status and diet quality. Particularly, non-white participants had markedly greater obesity rates than whites, and worse whole diet quality, with less whole grain, dairy, and fiber intake. The lower whole grain intake in this sample may explain most or all of the difference in fiber intake between whites and non-whites, as these are typically high fiber foods. Yet, limited but strong evidence exists identifying a similarly lower intake of both fiber and dairy in African Americans compared to whites (180–182). Overweight and obese participants also had lower SES in this sample, particularly those obese by WC for age. These results fall in line with previous studies, connecting lower SES (163,166–168) as well as African American (or nonwhite in the case of this sample) race (49,158–161) to poorer health outcomes, especially excess adiposity. However, it should be noted that the mean SES for the obese participants in this sample is not necessarily considered low for the country as a whole.

The very high degree of correlation between WC and BMI meets expectations. While BMI provides a very fast and minimally invasive representation of body fatness, it does not account for specific site accumulation of muscle, fat, or bone. As such, BMI tends to be less accurate in the very muscular and more densely boned. WC provides a more accurate representation of visceral fatness, a key indicator of increased inflammation and risk for chronic disease (31,126,131).

Unfortunately, this study is limited in its non-experimental design. As an observational study of these 16 year olds at one time point, no true conclusions can be drawn. Additionally, this study is limited in sample size at N=159, potentially limiting the ability to identify relationships among the dietary variables examined and BMI and WC. Also, while the sample was representative of the greater Greensboro, NC area at the time of recruitment, it is not representative of the state or country at large. The participants were almost exclusively white or African American, and the sample contains substantially more females than males. In addition, while BMI and WC are widely utilized and very useful tools, they are not the most accurate assessments of body fatness, and use of a Bod Pod or DXA machine would be preferable.

Conclusions

Although this research does not provide definitive evidence of a link between certain aspects of diet, it does add to limited body of evidence to demonstrate poor overall diet quality in the adolescent population, along with a fairly high rate of obesity. Additionally, this research implicates both excessive sweetened beverage intake and inadequate fruit intake as areas of particular concern for adolescents with implications for changes in adiposity, along with whole diet quality. Moreover, protein foods are identified as having the potential for a protective relationship against increases in adiposity. Lastly, this research highlights significant racial differences in both adiposity and diet composition, with higher rates of obesity by multiple measures and lower intakes of whole grains, fiber, and dairy products in non-whites.

This research also adds to a growing body of evidence that young Americans are relying on diets high in calories, sodium, and sugar dense foods, with poor intakes of nutrient dense items, especially those rich in fiber, such as whole grains, fruits, vegetables, and legumes, thus resulting in poor overall diet quality. Considering dietary habits adopted in adolescence have been linked to dietary habits in adulthood (6), long term poor diet quality originating in youth could result in an increase in the prevalence of obesity as well as a number of chronic health conditions, such as hypertension, cardiovascular disease, and type 2 diabetes mellitus. Future research should utilize experimental designs to identify interventions effective in improving diet quality to maximize long term health of the US population and decrease the risk for obesity and other chronic diseases.

Tables/Figures

Table 1. Sample Characteristics by Obesity Category

	Non-Obese BMI	Obese BMI	Non-Obese WC	Obese WC
Gender				
Male (n)	53	11	50	3
Female (n)	83	14	77	4
Race				
White (n)	95	11*	84	2*
Nonwhite (n)	41	14*	43	5*
SES	44.0 (14.1)	41.3 (16.6)	43.7 (14.4)	32.8 (15.0)

SES data expressed as mean (SD)
* Indicates significant difference at $p<0.05$

Table 2. Healthy Eating Index-2010 Scores of Obese and Non-Obese Adolescents by Body Mass Index (BMI) for Age

	Score Range	Total Sample	Non-Obese BMI	Obese BMI	Recommendation /1,000 kcal (max score)	Non-Obese Meeting Recommendation (%)	Obese Meeting Recommendation (%)
Total Fruit	0-5	1.5 (1.5)	1.5 (1.5)	1.1 (1.4)	≥ 0.8 c	5	4
Whole Fruit	0-5	1.5 (1.8)	1.5 (1.8)	1.4 (1.7)	≥ 0.4 c	9	8
Total Vegetables	0-5	2.7 (1.4)	2.7 (1.4)	2.7 (1.2)	≥1.1 c	12	4
Greens and Beans	0-5	1.6 (2.0)	1.7 (2.1)	1.3 (1.9)	≥ 0.4 c	18	16
Whole Grains	0-10	3.7 (3.0)	3.8 (3.0)	2.8 (2.8)	≥1.5 oz	7	4
Dairy	0-10	6.4 (3.1)	6.5 (3.1)	5.9 (3.1)	≥ 1.3 c	26	12
Total Protein Foods	0-5	4.4 (1.0)	4.3 (1.0)	4.7 (0.6)	≥ 2.5 oz	57	68
Seafood and Plant Proteins	0-5	1.7 (2.0)	1.7 (2.0)	1.6 (2.0)	≥ 0.8 oz	17	20
Fatty Acids	0-10	4.9 (3.1)	4.9 (3.2)	5.1 (2.6)	(MUFAs+PUFAs) / SFAs ≥ 2.5	10	4
Refined Grains	0-10	4.1 (3.4)	4.3 (3.5)	3.4 (3.0)	≤ 1.8 oz	9	0

Sodium	0-10	3.6 (2.9)	3.7 (3.0)	3.0 (2.8)	≤ 1.1 g	4	0
Empty Calories	0-20	13.1 (5.0)	13.3 (5.0)	12.3 (5.2)	≤ 19% of total kcal	12	12
Total HEI-2010 Score	0-100	49.2 (12.0)	50.0 (12.1)	45.2 (10.5)	≥ 80 (100)	1	0

Data expressed as mean (SD)

No significant differences observed at $p<0.05$ between Non-Obese and Obese by BMI

Table 3. Nutrient and Dietary Component Intake of Obese and Non-Obese Adolescents

Category	Non-Obese BMI	Obese BMI
Energy (kcal/d)	1799 (650)	1771 (608)
Trans Fat (g/d)	2.2 (1.6)	2.4 (1.4)
% Kcal from Fat	34.6 (6.3)	35.0 (5.5)
% Kcal from Sat Fat	11.3 (2.6)	11.3 (2.0)
Omega-3 Fatty Acids (g/d)	1.8 (0.9)	2.0 (1.1)
Fiber (g/d)	14.1 (7.1)	11.3 (4.2)
Added Sugars (g/d)	70.4 (47.0)	76.7(60.2)
Sweetened Beverages (c/d)	1.4* (1.5)	2.3* (2.9)

Data expressed as mean (SD)

* Indicates significant difference between groups at $p<0.05$

Table 4. Relationships of Healthy Index-2010 Score and Dietary Components with Body Mass Index (BMI) and Waist Circumference (WC)

Independent Variables	Dependent Variables	Beta	p value	R^2
Race (O nonwhite, 1 white)	BMI	-0.270	0.001	0.135
Gender (0 female, 1 male)		-0.041	0.594	
SES		-0.047	0.556	
Total Fruit (c)		-0.203	0.008	
Race (O nonwhite, 1 white)	WC	-0.212	0.016	0.125
Gender (0 female, 1 male)		0.289	0.002	
SES		-0.081	0.354	
Total Protein (oz eq.)		-0.230	0.013	

Note Race, Gender, and SES were forced entry to use as controls.

Table 5. Significant Correlations with Body Mass Index (BMI) and Waist Circumference (WC), with Pearson Correlation Coefficients (r)

	BMI (kg/m^2)	WC (cm)	Total Fruit (c/d)	Total Protein (oz. eq./d)	Dietary Fiber (g/d)
BMI		r = 0.886‡	r = -0.212*		r= -0.180*
WC			r= -0.187*	r = -0.217*	

All correlations are controlled for Gender, Race (white or non-white), and SES
* Indicates significant relationship at $p<0.05$
‡ Indicates significant relationship at $p<0.001$

CHAPTER IV

DIET QUALITY, BLOOD PRESSURE, AND BLOOD LIPIDS IN ADOLESCENTS

Introduction

Dyslipidemia is an emerging health concern among the youth of the United States, with approximately 20.3% of US 12-19 year olds having at least one abnormal lipid level (7). Dyslipidemia is even more prevalent among obese adolescents, as 42.9% of that group has at least one abnormal lipid value (7). Although incidence of hypertension in this population is much lower at 2.5-4.5%, pre-hypertension rates range from 10% to 15.7% (38,61–64). Taken altogether, these numbers reflect an increasing concern for the development of cardiovascular disease (CVD), at a young age. With prior work identifying signs of CVD in teenagers (8), the adolescent age group seems to be an ideal target population for prevention efforts and identifying key CVD risks.

Overweight and obesity among adolescents are related to alterations in lipid and glucose metabolism, and blood pressure regulation. In conjunction, these alterations represent a greatly increased risk for the metabolic syndrome, which is noted to correspond with chronic systemic inflammation. This inflammation, although considered stable, corresponds to atherosclerotic vascular changes, such as those discovered by Berenson et al (8). However, with appropriate medical and/or lifestyle intervention, these vascular changes are predominantly reversible at this stage of life (12,13). As such,

elevated adiposity, hypertension, and dyslipidemia are important indicators of a potentially reversible atherosclerotic process in the adolescent age group. Additionally, obesity, with its known contributions to CVD development and progression, can be considered both a CVD risk and predictor of additional CVD risk.

With increasing prevalence of overweight and obesity among the adolescent population (1), identifying risk for chronic disease among younger age groups has become a priority in preventive medicine and public health efforts. Considering tissue dissection and plaque evaluation is not a viable screening tool for CVD intervention, factors related to development and progression of CVD will be examined to identify risk for CVD. Of particular interest in this research are measures of adiposity, blood pressure (BP), and blood lipids. A good deal of research connects adiposity measures with abnormal blood lipid values in adolescent populations. In particular, trends among adolescents consistently reveal BMI and/or WC as positively related to TC, TG, and LDL, while negatively related to HDL (31,32,39–48,50–53). In addition, these same measures of adiposity are positively associated with SBP and/or DBP in the adolescent population (31,45,46,51,53).

A preponderance of evidence supports abnormally high TC, LDL cholesterol, and TG in the blood, or abnormally low HDL cholesterol in the blood as significant independent risks for heart disease (24,25,35–37,183). To that effect, annual testing for these blood lipids within the adult population has become standard care to monitor the progression of cardiovascular disease in the primary care setting. Among adolescents,

less evidence directly supports the impact of elevated blood lipids on atherosclerosis and cardiovascular disease progression. However, the AHA does supply recommendations for optimal and suboptimal blood lipid values among the childhood and adolescent population based on National Heart, Lung, and Blood Institute expert panel recommendations (35,183). Considered abnormally high, and therefore at greater risk for heart disease, are TC ≥200 mg/dL, LDL cholesterol ≥130 mg/dL, and TG ≥150 mg/dL. Considered abnormally low and therefore at greater risk for heart disease is HDL ≤35 mg/dL. However, it should be noted that LDL cholesterol <100 mg/dL and HDL cholesterol ≥60 mg/dL are considered optimal.

Another strong independent risk factor for cardiovascular disease is persistently elevated blood pressure. Hypertension, or elevated blood pressure, is highly related to atherosclerosis (8,54–57). Consistently elevated blood pressure leads to weakening and narrowing of blood vessels, potentially allowing more plaque buildup and blockage. Specifically, Berenson et al. (noted above) found correlations to plaque presence were strong with systolic blood pressure (r=0.55), but weak with diastolic blood pressure (r=0.22) (8). Hypertension is known to lead to stroke, myocardial infarction, and cardiac failure in the adult population (58). While these are not major occurrences among youths, hypertension in adolescence is highly predictive of hypertension in adulthood (59,60).

Still, in order to prevent development of these CVD risk indicators, the contributing antecedents must be better understood. The influence of poor lifestyle habits, particularly diet, has been studied thoroughly among the adult population. However, the

impact of poor diet on CVD risk in adolescence is not well established. Moreover, very limited data exist examining the impact of whole diet quality to CVD risk among the younger population.

Specific aspects of nutrition are considered contributors to CVD among the adult population. A number of single nutrients or clusters of nutrients have been implicated as contributors to elevated BP or dyslipidemia (14–18). Sugar, particularly in beverage form, has been examined by multiple researchers for its impact on adolescent weight, adiposity, and CVD risk. Relationships have been established between increasing sugary beverage or fructose intake and increasing BP, BMI, WC, and body weight in adolescents (87,119–126). Saturated fatty acid intake is also implicated in connection with elevated BP and TGs among adolescents (109,132), and total fat intake is related to elevated LDL (109). Dietary fiber intake, conversely, seems protective against CVD risks, as increasing fiber intake is related to decreasing visceral adiposity (130,131). Certain food groups, particularly dairy, fruit, vegetable, nuts, seeds, and legumes, are also negatively related to CVD risks in the forms of TC, LDL, and SBP (113,114,121,127–129). Lastly, sodium intake has been implicated with increasing SBP among adolescents (133).

Although single nutrient and food groups research has yielded insights into the connection between diet and CVD risk, a validated overall diet quality measure may be considered preferable, as it simultaneously encompasses multiple aspects of diet that may confound or offset the impact of specific nutrients or food groups. The Healthy Eating Index-2010 (HEI-2010) is a validated measure of total diet quality based on the 2010

Dietary Guidelines for Americans (19). Utilizing this measure, US adolescents have consistently poor diet quality, with recent reports identifying average scores between 46 and 50 out of a possible 100 (3–5,19,151,163). Minimal research exists connecting any variant of the HEI with CVD risks in the adolescent population, but some studies report a higher diet quality is related to decreasing adiposity and more favorable blood lipids (115,146,156). However, those studies that have examined the relationship between diet quality and CVD risk had samples that are not generalizable to the greater US population; they were either cancer survivors, low-income African-Americans or Middle Eastern adolescents.

While most CVD risk research has focused on middle to older age, the purpose of this study was to explore potential CVD risk factors in adolescence, a period in which early/initial stages of CVD may manifest. The aim of this study was to examine the relationships between diet quality and CVD risks in a group of 16 year-old adolescents. Another aim was to identify potential interactions between diet quality and adiposity in predicting multiple CVD risks. Recent literature suggests increased CVD risk in metabolically unhealthful lean adults, and low risk for metabolically healthful obese individuals (20–22). As such, examining the interaction of measures of adiposity and diet quality in predicting other CVD risks may provide unique insight into the presence (and development) of metabolically healthful obesity and/or metabolically unhealthful leanness.

Subjects and Methods

Sample

A subsample of adolescents participating in "The Right Track" ongoing research study was used for this dissertation research study. This study examined exclusively 16 year olds from this study, and 163 participants provided diet information in the form of dietary recalls.

The data obtained for this research occurred entirely within the "RIGHT Track" participants' 16th year of age, from February 2014 to October 2015.

More details on the sample can be found in the previous chapter.

Human Subjects Protections

This study has been approved by the Institutional Review Board of the University of North Carolina at Greensboro. All adolescent participants for this study provided assents to participate. All parents/guardians of the minor participants provided consent for the adolescent to participate.

Blood Pressure

Blood pressure is measured according to the National Heart, Lung, and Blood Institute standards (184). The participant is seated upright in a comfortable chair, and the research assistant measures by sphygmomanometer and stethoscope at the antecubital region. The participant is provided 5 minutes rest in the chair prior to first measurement,

and 5 minutes rest after the first measurement, prior to measuring a second time at the same site.

Diet and Anthropometrics

More detailed information on methodology for dietary information and anthropometric measurements can be found in the previous chapter and Appendix A.

Blood Lipids

For the venipuncture and blood draw, universal precautions were taken. The trained phlebotomist identified the venipuncture site according to the World Health Organization (WHO) guidelines for site selection. The trained phlebotomist also followed Occupation Safety and Health Administration guidelines for blood handling.

The trained phlebotomist began by explaining the blood draw procedures, then performed the collection of a maximum of 20 mL whole blood. Blood was drawn into SST Vacutainer® type tubes (aka Tiger Tops), which were de-identified, containing subject identification number only. These tubes were gently inverted approximately ten times, then placed upright at room temperature for 20 minutes to allow for clotting. Next, the tubes were centrifuged for 20 minutes at 3000 rpm at 4°C. Serum, then separated, and pipetted from SST Vacutainer® type tubes into microtubes in 500 microliter aliquots, labeled with subject ID#, and stored at -80 °C until ready to be analyzed. Colorimetric assay was used to measure, total, HDL, and LDL cholesterol, and triglycerides using microtiter plate procedures outlined by the company (Wako Chemical, Richmond, VA).

Statistics

SPSS Statistics (Microsoft, version 20) was used to perform all statistical analyses. Obese was defined as BMI $\geq 95^{th}$percentile for age, and abdominally obese as WC $\geq 90^{th}$percentile for age based on NHANES data. Participant data was grouped based on categorical cutoffs identified for CVD risk, and classified as obese (BMI $\geq 95^{th}$percentile for age) vs. non-obese; abdominally obese (WC $\geq 90^{th}$percentile for age) vs. non-obese; elevated BP (BP $\geq 90^{th}$percentile for age) vs. normal BP; abnormal blood lipids (TC $\geq$ 200 mg/dL, LDL $\geq$ 130mg/dL, TG $\geq$ 150 mg/dL, or HDL $\leq$ 35 mg/dL) vs. normal blood lipids.

Descriptive statistics were determined for the sample, including mean and standard deviation for SES, BMI, WC, SBP, DBP, HDL, LDL, TC, and TG. Next, ANOVA was used to compare means across all the variables noted above between hypertensive and normotensive participants, and between dyslipidemic participants and those with normal blood lipids. Additionally, ANOVA was run to compare means of SBP, DBP, TC, LDL, HDL, TC and TG across genders, obesity by BMI for age and non-obese, and obesity by WC for age and non-obese.

Next, ANOVA was run comparing the means of HEI-2010 score and each HEI component score (total fruit, whole fruit, total vegetable, greens and beans, whole grains, dairy, total protein, seafood and plant protein, fatty acids, refined grains, sodium, and empty calories) between hypertensive and normotensive participants, and dyslipidemic participants and those with normal blood lipids. The researcher also performed ANOVA

between hypertensive and normotensive participants to compare mean intakes of energy, trans fats, percentage of energy from fat, percentage of energy from saturated fat, omega 3 fatty acids, dietary fiber, added sugars, sweetened beverages, sodium, calcium, magnesium, and potassium. Similarly, ANOVAs were run to compare means of all the same variables (excluding the mineral intakes) between participants with low and normal HDL, high and normal LDL, high and normal TC, and high and normal TG. Differences for all ANOVAs were considered significant at $p<0.05$.

The researcher also ran a correlation table among all variables (SBP, DBP, HDL, LDL, TC, TG, BMI, WC, total fruit, whole fruit, total vegetable, greens and beans, whole grains, refined grains, total proteins, seafood and plant proteins, empty calories, dairy, energy, percentage of energy from fat, percentage of energy from saturated fat, omega 3 fatty acids, dietary fiber, added sugars, sweetened beverages, sodium, potassium, magnesium, calcium, and HEI score) to identify multicollinearity, and significant correlations between the variables. Sweetened beverages included beverages with added sugar such as soda, lemonade, kool aid, sweet tea, sports drinks, and sweetened coffee drinks, but excluded fruit juice and flavored milk. The researcher then ran eight regression analysis of HEI-2010 scores with all CVD risk variables: BMI, WC, SBP, DBP, TC, TG, LDL, and HDL as outcome variables. Correlations between HEI-2010 and any variable were considered significant at $p<0.05$. Race, gender, and SES were considered as covariates in the regression analyses.

The researcher ran hierarchical regression analyses. Dependent variables included BP and blood lipids. Independent variables included BMI, WC, HEI-2010 score, race, SES, and gender. Additionally, interaction terms of HEI-2010*BMI and HEI-2010*WC were entered as independent variables. HEI-2010, BMI and WC were centered prior to multiplying the terms. Any independent variable in the model was considered significant at $p<0.05$. Impact and nature of interaction effects were examined using graphical representation.

More specifically, two models were run for both theoretical and practical applications. Because we anticipated a high collinearity among BMI and WC, we ran hierarchical regression analyses including both BMI and WC. Thus, one model began with entry of BMI, then entry of HEI, then entry of HEI-2010*BMI, and finally with forced entry of the covariates race, SES and gender. The other model began with entry of WC, then entry of HEI-2010, then entry of HEI-2010*WC, and finally forced entry of race, SES, and gender.

Lastly, we ran regression analysis of TC, TG, LDL, and HDL with total fat, saturated fat, omega-3 fatty acid, refined grain, whole grain, fruit, vegetable, fiber, and added sugar intakes. Additionally, we ran regression analyses of BP with total fat, saturated fat, sodium, fruit, vegetable, fiber, potassium, magnesium, calcium, omega-3 fatty acid, and added sugar intakes. Correlations were considered significant at the $p<0.05$ level.

Results

A total of 163 adolescents agreed to participate in diet recalls. One hundred forty nine participants provided three days of recall, six provided two days of recalls and eight provided only one day of recalls. There were no significant differences in the average HEI scores nor any other dietary variables analyzed between these three groups. One hundred forty three participants provided anthropometric data by coming to the UNCG Exercise Physiology Lab for measurements. An additional 19 self-reported height and weight data either due to excessive distance to travel to the lab or scheduling conflicts. It should be noted that correlations between self-reported BMI (from the provided height and weight) and BMI determined from height and weight measurements taken at the lab were very strong ($R=0.961$, $p<0.001$). One hundred forty three participants provided blood pressure data at the lab, and 97 participants agreed to provide blood samples. As such, the total sample size for BP analyses was 143, while the sample size for lipid analyses was 97.

Table 6 displays descriptive statistics about the CVD risks analyzed in this research. Table 6 also demonstrates differences in those considered hypertensive or not, or dyslipidemic (at least one blood lipid value not in line with AHA recommendations) or not by gender, race, SES, BMI, WC, BP, and blood lipids. No significant differences were observed in rates of hypertension between genders, races, or by SES. Hypertensive participants had significantly higher BMI, WC, SBP, DBP, and TG compared to non-hypertensive. No significant differences were observed in rates of dyslipidemia by

gender, race, or SES. Dyslipidemic participants had significantly higher LDL and TC than those with normal lipids, but no differences in HDL or TG. Additionally, no difference was observed in BMI between participants with dyslipidemia or not, while participants with dyslipidemia had significantly lower WC than those with normal blood lipids. Further analysis of classification for CVD risk, based on the TC:HDL ratio, identified 29.1% of adolescents considered in the above average risk for CVD (≥4.5 for females, ≥5.0 for males).

Table 7 highlights differences in BP and blood lipids by gender and obesity category. SBP and DBP are both significantly higher in both obese groups (by BMI and WC) compared to non-obese groups. However, the only significant difference observed in blood lipids by obesity status reveals a lower TC among those obese by BMI compared to non-obese.

Table 8a reflects the differences observed in HEI scores among the total sample, hypertensive participants, and non-hypertensive participants. One significant difference was observed in the scores of each component, with no differences observed in the rate of participants meeting the recommendation for each component. Participants with hypertension had significantly higher scores in the total protein foods category compared to non-hypertensive participants. Moreover, no significant differences were observed in total HEI scores or rate of participants meeting recommendations for total HEI score among hypertensive and non-hypertensive participants.

Table 8b shows differences observed in HEI scores among the total sample, dyslipidemic participants, and participants with normal blood lipids. No differences were observed in HEI component scores between dyslipidemic participants and those with normal blood lipids. However, rate of dyslipidemic participants meeting the recommendation for greens and beans intake was significantly lower than those with normal blood lipids at 13% vs. 33%.

As shown in Table 9a, hypertensive participants had significantly lower energy and trans fat intake than non-hypertensive participants. Displayed in Table 9b, a similar difference in energy intake was observed in participants with high total cholesterol compared to participants with normal TC. Additionally, participants with normal TC ate significantly more omega-3 fatty acids and dietary fiber compared to those with high TC. Primary food source of omega-3 fatty acids among the sample was soybean oil, most often as a primary ingredient in a salad dressing or pre-prepared food item. Differences were also observed in diet between those with normal or elevated LDL and TG. Participants with elevated LDL consumed more sweetened beverages (note this measure excluded fruit juice and flavored milk) than those with normal LDL. However, participants with high TG consumed fewer sweetened beverages and added sugars than participants with normal TG.

Table 10 highlights significant correlations between BP, blood lipids, and a number of dietary components. In particular, SBP was significantly positively correlated with DBP, BMI, and WC, and negatively correlated with total vegetables, dietary fiber,

and magnesium. Additionally, DBP was significantly positively correlated with BMI, WC, and % Kcal from total fat, and negatively correlated with total fruit, whole fruit, greens and beans, and dietary fiber. HDL cholesterol was significantly negatively correlated with LDL cholesterol, but positively correlated with TG. LDL cholesterol was significantly positively correlated with TC, and negatively correlated with total vegetables as well as greens and beans. TC was significantly negatively correlated with WC, total vegetables, greens and beans, omega-3 fatty acids, dietary fiber, potassium, magnesium, and HEI score. Lastly, TG was not significantly correlated to any additional dietary components.

Table 11 displays only the significant results of linear regression analyses between BP, blood lipids, and dietary components controlled with forced entry of gender, race, and SES. In the model for SBP, the variables accounted for only 7.7% of the variance, with total vegetables being the only significant variable. In the model for DBP, the variables accounted for 9.8% of the variance, with dietary fiber and gender the only significant variables. Variables in models for HDL and LDL explained a similar amount of variance, at 10.3% and 10.0%, respectively. Whole grains and SES were considered significant in the model for HDL, while greens and beans was the only significant variable in the model for LDL. The model for TC contained four significant variables which accounted for 18.7% of the variance. SES, greens and beans, omega-3 fatty acids, and refined grains were all considered significant in the model. By contrast, race and dietary fiber were the only significant variables in the model for TG, which explained 12.2% of the variance.

Table 12 displays potential interaction effects between BMI, WC, and diet quality on BP and blood lipids. Both models for SBP were considered significant. In the first, BMI and the interaction term of BMI and HEI were both considered significant. BMI explained 17.5% of the variance in the model, while the interaction term explained an additional 5.4% toward the total model, which accounted for 23.8% of the variance. In the second model, which explained 18.4% of the variance, WC was the only variable considered significant and explained 17.0% of the variance alone. Both models for DBP were also considered significant. In the first, BMI and gender were significant variables, adding 11.7% and 2.4% to the total 14.5% of the variance accounted for by the model. In the second, WC was again the only significant variable in the model, accounting for 11.7% of the 16.0% of variance explained by the model. Neither model for HDL were considered significant. However, in the second model (containing WC as an independent variable), SES was considered a significant variable and accounted for 5.4% of the variance. Neither model for LDL was considered significant, nor did either contain a significant variable. Both models for TC were significant and contained multiple significant variables. In the first model for TC, BMI, HEI, gender, and SES were considered significant. The total model accounted for 20.3% of the variance, with BMI accounting for 3.1%, HEI accounting for 7.2%, gender accounting for 4.6%, and SES accounting for 4.4%. However, BMI was only considered significant after HEI was entered. In the second model for TC, WC, HEI, and SES were considered significant. The model explained 18.3% of the variance. WC explained 4.6%, HEI explained 6.2%, and SES explained 4.2% of the variance. Neither model for TG was considered significant.

However, in the first model (including BMI as an independent variable), the interaction term of BMI and HEI was considered significant, and explained 4.6% of the variance.

Figure 1 and Figure 2 display graphical representation of the interaction effects of BMI and HEI on SBP and TG, respectively. In Figure 1, lowest SBP was observed among participants with low BMI and high HEI, while highest SBP was observed in participants high BMI and high HEI. A very similar trend was observed in Figure 2, as TG was lower among participants with low BMI and high HEI compared to average or high HEI, while TG was higher among participants with high BMI and high HEI compared to average or low HEI.

Discussion

Rates of both hypertension and dyslipidemia in this sample were higher than those seen in other adolescent research. Compared to other US adolescent studies showing rates of HTN in the 2.5-4.5% (38,61–64), the rate in this sample was more than double at 10.6%. This may be a common characteristic of the region of the US in which participants were sampled. Previous work in adults has identified higher SBP and DBP, and greater incidence of HTN in the southeastern US, also known as the "stoke belt" (185,186). Additionally, this sample had a much higher rate of participants with at least one abnormal blood lipid value at 72.2%, compared to the 20.3% rate found in a 2010 CDC report and the 42.9% rate among obese adolescents found in the same report (7). More specifically, 51.5% of this sample had elevated TC. This strikingly high rate of elevated TC may be misleading among this sample, however, as mean HDL was over 60

mg/dL, creating a scenario in which many dyslipidemic participants do not have excessively high non-HDL cholesterol. In particular, at age 16, continuing growth, certain medication use (particularly birth control), and lack of exposure to substances such as cigarette smoke and androgens may favor higher HDL concentrations (187,188). Upon further analysis, 70.1% of the sample had TC:HDL ratios predictive of below average heart disease risk (among adults), at <4.5 for females and <5.0 for males (189). These factors suggest CVD risk related to blood lipids among this sample may be more similar to national average, noted by Kit et al near 20% in children aged 8-17 years (190), as measured by TC:HDL ratio. These discrepancies also reflect the substantive limitation of any single blood lipid marker in interpreting CVD risk compared to the entirety of the blood lipid profile.

Findings in this research relating increases in adiposity (both BMI and WC) with increases in BP agree with previous findings in adolescents (31,45,46,51,53). Increases in both BMI and WC related to increases in SBP and DBP, obese participants had higher SBP and DBP, and hypertensive participants had higher BMI and WC. Additionally, hypertensive participants had higher TG, which agrees with previous research (191) and fits two of the key classifications of the metabolic syndrome (192).

By contrast, the findings of this research conflict with multiple studies establishing a relationship between increases in BMI and WC with increases in TC, LDL, and TG, and decreases in HDL (31,32,39–48,50–53). This research instead suggests lower TC among obese participants, and a decrease in TC with increases in BMI and

WC, with no relationships between adiposity and LDL, HDL, or TG. This conflict may be related to differences in fitness across BMI and WC categories. As BMI and WC are not specific measures of body composition, a significant number of the participants may have higher than normal lean body mass, possibly associated with athletics and higher fitness. In that case, higher fitness may be creating a cholesterol-lowering effect, as has been observed in prior research (193–197). Furthermore, the accuracy of BMI as a body composition for African Americans has been questioned by multiple researchers (198,199), as African Americans commonly have higher bone density and more muscle at a given height and weight compared to Caucasians. Additionally, higher prevalence of birth control use among female participants with lower BMI may alter these results, as birth control tends to increase total cholesterol (200,201). Lastly, recent weight loss tends to cause significant declines in total cholesterol, even if a normal BMI has not been achieved (202–204). Obese participants in this study may have made efforts to lose weight prior to blood draw, leading to lower total cholesterol despite not yet achieving a normal BMI for age.

The high correlations observed between most CVD risk measures meet expectations. As identified previously in the adult literature (205,206), SBP and DBP are highly positively related. Additionally, as previously identified in the Framingham Study and fitting intuitive expectations of blood lipid biochemistry (207), increases in LDL related to increases in TC, and decreases in HDL. Contrary to the findings of the Framingham study where HDL and TG were negatively related, HDL in this research

was positively related to TG. This may be related to contraceptive use among female participants, as contraceptives tend to raise TC, LDL, HDL, and TG (200,201).

Many of the relationships between diet and CVD risks identified in this study reflect that seen in previous research in adolescents (14–18,113–115,121,127–129,146,156,208). In particular, a variety of aspects of the DASH diet are associated with lower SBP and DBP. Greater intakes of total vegetables and dietary fiber are associated with lower SBP, and greater intakes of total fruit, whole fruit, greens and beans, and dietary fiber, as well as lower intake of total fat are related to DBP. Greater intake of magnesium was also identified as related to lower SBP. Similar diet patterns were observed in this study in relation to blood lipids. A pattern of greater intake of greens and beans, dietary fiber, omega 3 fatty acids, and total vegetables, lower refined grain and sweetened beverage intake, and a higher HEI score was related to lower TC and LDL.

A number of relationships observed in this study conflict with expectations based on prior literature. Energy and trans fat intake was lower among participants with HTN. Among participants with HTN, protein intake was higher in this study, which was not observed in previous research. Lastly, this research did not identify relationships between BP and sodium (133), potassium (97,99,100), or saturated fatty acids (109,132), which was likely a function of consistently high sodium and saturated fat, with low potassium intake among this sample. In direct contrast to expectations (109), higher TC was related to lower intake of energy and trans fat. In a similar vein, higher TG was related to fewer sweetened beverages and added sugars, and higher HDL was related to lower whole grain

intake. These unexpected findings regarding energy intake and HTN or high TC may be related to an under-reporting issue among the obese participants in this sample. Under-reporting of energy intake among the obese is a common issue noted in previous research (176–179), and obese participants in this sample had significantly higher BP. These relationships may otherwise be related to participation in athletics, with reliance on and regular consumption of sweetened sports drinks and low fiber carbohydrate foods as snacks before, during, or after practice. Athletes making these choices would likely have lower TG and higher HDL, and tend to eat more carbohydrate, less protein. Additionally, participants engaging in athletics or regular exercise would likely eat more calories to compensate for the energy used in activity, making energy intake higher compared to sedentary participants. Also, a negative relationship was observed between TC with magnesium and potassium, which is not noted in previous research. This may reflect the potassium content of most vegetables and magnesium content of a variety of plant and dairy foods like nuts, seeds, and milk. This research did not identify a relationship between any blood lipids and saturated fats, which are consistently implicated in increases to TC, LDL, and TG in the adult literature (25,52,109–111), likely related to the high saturated fat intake across the entire sample.

Interaction effects from BMI and diet quality were identified in this research that have previously been unexamined. Upon graphical representation of these effects, better diet quality has an enhancing effect on CVD risk reduction at lower than average BMI, but a deleterious effect at higher BMI, leading to increased SBP and TG. These findings are at least partially contrary to the anticipated protective effects of better diet quality at

all levels of BMI. These unexpected results may be related to participants with a history of overweight or obesity attempting to improve diet, but not achieving significant weight loss, and not achieving significant decreases to SBP or TG. This pattern may also be reflective of a poorer eating pattern among athletes with higher BMI compared to sedentary participants with higher BMI, and the CVD risk reduction associated with higher aerobic fitness. Regardless, moderation effects of HEI on the relationships between BMI and SBP and TG in adolescents were novel findings.

Despite some conflicts between the findings in this research and previous research, this research presents a number of strengths. This study utilized gold standard methodology for collecting diet information, BP, and blood lipids, among a sample of socioeconomically diverse group of 16 year olds. Additionally, this study examined variables not commonly explored in this population, particularly BP, blood lipids, whole diet quality, and the interactions between them. However, this research is limited in its observational design and relatively small sample size reflecting almost exclusively white and African American participants. As such, no cause and effect pattern can be established, and the results may not be generalizable to the whole US adolescent population. Moreover, body composition using BMI does not necessarily reflect obesity among those with greater lean body mass. Dual-energy absorptiometry, BodPod, or underwater weighing would be accurate methods to measure body fatness. Lastly, aerobic fitness is considered one of the best predictors of CVD risk, but was not measured in this study. VO_2 max testing would be ideal to measure fitness level, in addition to use of accelerometers to track physical activity.

While this study cannot provide a causal link between aspects of diet and HTN or dyslipidemia in the adolescent population, a number of key protective factors emerge that concur with the limited research among this age group. Particularly, greater intakes of total vegetables, total fruit, whole fruit, greens and beans, dietary fiber, and magnesium are implicated with lower SBP and DBP. In contrast, higher proportion of calories from fat potentially predicts higher DBP. Also, higher BMI and WC are highlighted as greater risks for elevated SBP and DBP. Additionally, total vegetables, greens and beans, and dietary fiber show protective effects against elevated TC and LDL. Omega 3 fatty acids and HEI additionally emerge in this research as potentially predictive of lower TC. Lastly, simple carbohydrate intake, in the forms of sweetened beverages and refined grains, is implicated in higher LDL and TC.

This research adds to a limited body of evidence connecting diet to both dyslipidemia and HTN at a young age. Notably, the protective effects of many aspects of diet, particularly higher fiber plant foods, are more consistently connected to BP and blood lipids than the negative impacts of other aspects of diet (14–18,113,114,121,127–129,208). Additionally, this study confirmed the importance of appropriate body composition in HTN prevention (31,45,46,51,53). Yet, this research reflects the potential positive impact of greater intake of plant foods rich in fiber, particularly vegetables, fruits, and legumes on CVD risk reduction at a young age, as well as the negative impact of sweetened beverages and refined grains on CVD risk. Lastly, this research has revealed moderation effects of HEI on the relationships between BMI and CVD risks. Future research should utilize experimental designs to analyze the full impact on

restriction of simple carbohydrate and greater inclusion of high fiber plant foods to elucidate the potential impacts of relatively small alterations to diet on reduction of CVD risk from a young age. Larger scale observational research should explore the moderation of the connection between body composition and CVD risk using more accurate methods for assessing body fatness. Finally, interactions effects of aerobic fitness on relationships between diet and CVD risk should be examined for enhanced or diminished CVD risk reduction via diet improvement.

Tables/Figures

Table 6. Sample Characteristics by Cardiovascular Disease Risk Category

	Total Sample	Non-Hypertensive	Hypertensive	Normal Blood Lipids	Dyslipidemic
Gender					
Male (n)	65 (40%)	50 (91%)	5 (9%)	14 (34%)	27 (66%)
Female (n)	97 (60%)	77 (89%)	10 (11%)	13 (23%)	43 (77%)
Race					
White (n)	106 (65%)	83 (89%)	10 (11%)	16 (27%)	44 (73%)
Nonwhite (n)	56 (35%)	44 (90%)	5 (10%)	11 (30%)	26 (70%)
Socioeconomic Status (SES)	43.5 (14.4)	43.7 (14.3)	40.4 (16.0)	43.1 (15.5)	44.9 (12.5)
Body Mass Index (BMI) (kg/m^2)	24.5 (6.3)	23.7‡ (5.4)	28.5‡ (8.7)	25.9 (7.5)	23.8 (5.2)
Waist Circumference (WC) (cm)	78.6 (14.2)	77.5‡ (12.7)	88.3‡ (22.0)	84.7* (20.7)	77.0* (11.3)

Systolic Blood Pressure (SBP) (mmHg)	113.7 (9.9)	111.8‡ (8.3)	129.7‡ (7.6)	114.5 (9.7)	113.1 (9.6)
Diastolic Blood Pressure (DBP) (mmHg)	68.9 (9.1)	67.7‡ (8.2)	78.9‡ (10.5)	68.6 (8.3)	68.6 (9.8)
High Density Lipoprotein (HDL) (mg/dL)	62.9 (21.7)	63.0 (21.5)	61.8 (23.8)	66.6 (10.6)	61.4 (24.5)
Low Density Lipoprotein (LDL) (mg/dL)	107.0 (38.5)	105.3 (37.6)	121.8 (45.4)	80.6‡ (17.5)	117.2‡ (39.6)
Total Cholesterol (TC) (mg/dL)	209.9 (45.3)	209.1 (44.1)	216.8 (57.1)	172.7‡ (19.3)	224.2‡ (44.3)
Triglycerides (TG) (mg/dL)	118.0 (48.8)	113.7‡ (45.8)	155.3‡ (60.6)	114 (34.4)	119.5 (53.5)

SES, BMI, WC, SBP, DBP, HDL, LDL, TC, and TG data expressed as mean (SD)
SES measured by Hollingshead score, with a possible range of 8-66
* Indicates significant difference at $p<0.05$
‡ Indicates significant difference at $p<0.01$

Table 7. Blood Pressure and Blood Lipids by Obesity Category

	Male	Female	Non-Obese Body Mass Index	Obese Body Mass Index	Non-Obese Waist Circumference	Obese Waist Circumference
Systolic Blood Pressure (mmHg)	114.6 (8.4)	113.2 (10.7)	112.6‡ (9.6)	119.8‡ (9.2)	112.6‡ (9.7)	125.6‡ (8.3)
Diastolic Blood Pressure (mmHg)	70.7 (9.3)	67.8 (8.8)	67.7‡ (8.9)	75.6‡ (7.3)	68.3‡ (9.1)	78.2‡ (2.5)
Total Cholesterol (mg/dL)	201.1 (51.2)	216.3 (38.9)	214.0* (47.3)	187.3* (21.5)	211.4 (46.6)	185.2 (20.6)
Low Density Lipoprotein (mg/dL)	100.8 (38.8)	111.5 (38.0)	107.4 (39.7)	104.5 (32.4)	107.6 (39.4)	98.5 (31.0)
High Density	62.3	63.3	62.6	64.4	62.3	65.6

Lipoprotein (mg/dL)	(20.2)	(22.8)	(22.5)	(17.1)	(22.2)	(14.2)
Triglycerides (mg/dL)	112.5 (44.1)	122.0 (52.1)	119.5 (50.7)	109.3 (37.4)	118.4 (49.8)	96.0 (35.2)

Data expressed as mean (SD)

* Indicates significant difference at $p<0.05$

‡ Indicates significant difference at $p<0.01$

Table 8a. Healthy Eating Index-2010 (HEI-2010) Scores of Hypertensive (HTN) and Normal Blood Pressure (Non-HTN) Adolescents

	Score Range	Total Sample	Non-HTN	HTN	Recommendation /1,000 kcal (max score)	Non-HTN Meeting Recommendation (%)	HTN Meeting Recommendation (%)
Total Fruit	0-5	1.5 (1.5)	1.5 (1.5)	1.1 (1.1)	≥ 0.8 c	5	0
Whole Fruit	0-5	1.5 (1.8)	1.4 (1.7)	1.8 (2.0)	≥ 0.4 c	9	7
Total Vegetables	0-5	2.7 (1.3)	2.8 (1.4)	2.3 (1.0)	≥1.1 c	11	0
Greens and Beans	0-5	1.7 (2.0)	1.7 (2.0)	1.3 (1.9)	≥ 0.4 c	18	13
Whole Grains	0-10	3.6 (3.0)	3.5 (3.0)	4.4 (3.5)	≥1.5 oz	6	13
Dairy	0-10	6.5 (3.0)	6.4 (3.1)	7.5 (2.7)	≥ 1.3 c	22	40
Total Protein Foods	0-5	4.4 (1.0)	4.3* (1.0)	4.9* (0.2)	≥ 2.5 oz	57	67
Seafood and Plant Proteins	0-5	1.7 (2.0)	1.8 (2.0)	1.6 (2.0)	≥ 0.8 oz	17	20
Fatty Acids	0-10	4.9 (3.0)	4.8 (3.1)	5.1 (3.0)	(MUFAs+PUFAs) / SFAs ≥ 2.5	9	0

Refined Grains	0-10	4.1 (3.4)	4.1 (3.5)	4.3 (3.0)	≤ 1.8 oz	7	7
Sodium	0-10	3.5 (2.9)	3.6 (2.9)	2.9 (2.6)	≤ 1.1 g	2	0
Empty Calories	0-20	13.3 (4.7)	13.2 (4.8)	14.4 (3.8)	≤ 19% of total kcal	11	13
Total HEI-2010 Score	0-100	49.4 (11.8)	49.1 (11.8)	51.7 (11.8)	≥ 80 (100)	2	0

Data expressed as mean (SD)

* Indicates significant difference at $p<0.05$

Table 8b. Healthy Eating Index-2010 (HEI-2010) Scores of Dyslipidemic and Adolescents with Normal Blood Lipids

	Score Range	Total Sample	Normal Blood Lipids	Dyslipi-demic	Recommendation /1,000 kcal (max score)	Normal Blood Lipids Meeting Recommendation (%)	Dyslipidemic Meeting Recommendation (%)
Total Fruit	0-5	1.5 (1.5)	1.7 (1.7)	1.3 (1.4)	≥ 0.8 c	7	1
Whole Fruit	0-5	1.5 (1.8)	1.6 (1.9)	1.3 (1.7)	≥ 0.4 c	15	4
Total Vegetables	0-5	2.7 (1.3)	2.8 (1.2)	2.7 (1.4)	≥1.1 c	7	13
Greens and Beans	0-5	1.7 (2.0)	2.3 (2.3)	1.4 (1.9)	≥ 0.4 c	33*	13*
Whole Grains	0-10	3.6 (3.0)	3.2 (2.5)	3.3 (3.0)	≥1.5 oz	4	6
Dairy	0-10	6.5 (3.0)	7.1 (2.6)	6.0 (3.2)	≥ 1.3 c	26	20
Total Protein Foods	0-5	4.4 (1.0)	4.4 (1.0)	4.4 (1.0)	≥ 2.5 oz	63	59

Seafood and Plant Proteins	0-5	1.7 (2.0)	1.7 (1.9)	1.5 (2.0)	≥ 0.8 oz	11	16
Fatty Acids	0-10	4.9 (3.0)	4.6 (2.9)	4.9 (3.1)	(MUFAs+PUFAs) / SFAs ≥ 2.5	7	9
Refined Grains	0-10	4.1 (3.4)	4.1 (3.0)	4.0 (3.4)	≤ 1.8 oz	7	7
Sodium	0-10	3.5 (2.9)	4.1 (3.2)	3.4 (2.7)	≤ 1.1 g	0	3
Empty Calories	0-20	13.3 (4.7)	13.1 (4.8)	13.3 (4.9)	≤ 19% of total kcal	4	11
Total HEI-2010 Score	0-100	49.4 (11.8)	50.6 (9.2)	47.4 (10.8)	≥ 80 (100)	0	0

Data expressed as mean (SD)

* Indicates significant difference at $p<0.05$

Table 9a. Nutrient and Dietary Component Intake of Hypertensive (HTN) and Non-Hypertensive (Non-HTN) Adolescents

Category	Non-HTN	HTN
Energy (kcal/d)	1847* (668)	1463* (521)
Trans Fat (g/d)	2.3* (1.6)	1.3* (0.6)
% Kcal from Total Fat	35.0 (6.1)	33.5 (7.1)
% Kcal from Saturated Fat	11.4 (2.5)	10.9 (2.2)
Omega-3 Fatty Acids (g/d)	1.9 (1.0)	1.6 (1.2)
Fiber (g/d)	14.0 (6.9)	11.0 (4.3)
Added Sugars (g/d)	72.0 (46.4)	55.5 (29.2)
Sweetened Beverages (c/d)	1.6 (1.8)	1.2 (1.1)

Sodium (mg/d)	3176 (1354)	2618 (1001)
Calcium (mg/d)	897 (420)	748 (338)
Magnesium (mg/d)	219 (86)	182 (57)
Potassium (mg/d)	1981 (805)	1565 (591)

Data expressed as mean (SD)
* Indicates significant difference between groups at $p<0.05$

Table 9b. Nutrient and Dietary Component Intake of Dyslipidemic and Adolescents with Normal Blood Lipids: High Density Lipoprotein (HDL), Low Density Lipoprotein (LDL), Total Cholesterol (TC), and Triglycerides (TG)

Category	Normal HDL	Low HDL	Normal LDL	High LDL	Normal TC	High TC	Normal TG	High TG
Energy (kcal/d)	1762 (631)	1934 (820)	1798 (648)	1767 (725)	1943* (787)	1646* (488)	1819 (660)	1703 (682)
Trans Fat (g/d)	2.0 (1.2)	2.5 (1.9)	2.1 (1.3)	2.2 (1.6)	2.2 (1.5)	2.0 (1.2)	2.1 (1.4)	2.1 (1.3)
% Kcal from Total Fat	34.9 (6.9)	33.3 (5.0)	35.1 (7.0)	33.4 (5.3)	34.6 (7.6)	34.7 (5.6)	34.3 (5.7)	35.6 (9.0)
% Kcal from Saturated Fat	11.3 (2.6)	11.3 (2.3)	11.3 (2.6)	11.1 (2.3)	11.1 (2.2)	11.4 (2.8)	11.2 (2.3)	11.6 (3.0)
Omega-3 Fatty Acids (g/d)	1.8 (1.0)	1.8 (1.0)	1.9 (0.9)	1.7 (1.0)	2.1** (1.1)	1.5** (0.7)	1.9 (1.0)	1.6 (0.9)
Fiber (g/d)	12.6 (5.6)	14.6 (7.9)	13.4 (6.5)	11.2 (4.3)	14.1* (6.6)	11.7* (5.3)	12.9 (6.3)	12.7 (5.5)
Added Sugars (g/d)	74.1 (48.6)	74.2 (44.0)	69.9 (38.9)	87.1 (67.2)	83.4 (54.8)	65.3 (38.3)	79.7* (50.5)	57.2* (33.2)
Sweetened Beverages (c/d)	1.8 (2.0)	2.0 (1.4)	1.6* (1.3)	2.5* (3.0)	2.1 (2.4)	1.5 (1.3)	2.0* (2.1)	1.0* (1.0)

Data expressed as mean (SD)
* Indicates significant difference between groups at $p<0.05$
** Indicates significant difference between groups at $p<0.01$

Table 10. Correlations to Blood Pressure, Blood Lipids, with Pearson Correlation Coefficients (r)

	Systolic Blood Pressure (SBP)	Diastolic Blood Pressure (DBP)	High Density Lipoprotein (HDL)	Low Density Lipoprotein (LDL)	Total Cholesterol (TC)	Triglycerides (TG)
SBP (mm Hg)						
DBP (mm Hg)	r = 0.519‡					
HDL (g/dL)						
LDL (g/dL)			r = -0.426‡			
TC (g/dL)				r = 0.574‡		
TG (g/dL)			r = 0.522*	r = -0.175		
BMI (kg/m^2)	r = 0.418‡	r = 0.343‡			r = -0.176	
WC (cm)	r = 0.412‡	r = 0.342‡			r = -0.215*	
Total Fruit (c)		r = -0.209*				
Whole Fruit (c)		r = -0.240**				
Total Vegetables (c)	r = 0.202*	r = -0.155		r = -0.236*	r = -0.220*	
Greens and Beans (c)	r = -0.164	r = -0.175*		r = -0.248*	r = -0.267**	
Whole Grains (oz eq)			r = -0.180			
Refined Grains (oz eq)	r = -0.143					

Total Proteins (oz eq)					r = -0.170	r = -0.173
Seafood and Plant Proteins (oz eq)		r = -0.164	r = -0.189			
Energy (kcal)						r = -0.172
Total Fat (% kcal)		r = 0.184*				
Saturated Fat (% kcal)		r = 0.157				
Omega-3 Fatty Acids (g)					r = -0.227*	
Dietary Fiber (g)	r = -0.203*	r = -0.203*			r = -0.214*	
Added Sugars (g)				r = 0.170		
Sweetened Beverages (c)				r = 0.172		
Sodium (mg)			r = -0.188			
Potassium (mg)					r = -0.236*	
Magnesium (mg)	r = -0.172*	r = -0.141			r = -0.213*	
Calcium (mg)	r = -0.151					
Healthy Eating Index 2010 score		r = -0.160		r = -0.179	r = -0.237*	

Only those with $p \leq 0.10$ are reported.
* Indicates significant relationship at $p<0.05$
** Indicates significant relationship at $p<0.01$
‡ Indicates significant relationship at $p<0.001$

Table 11. Relationships of Healthy Eating Index-2010 and Diet Components with Blood Pressure, Blood Lipids

Independent Variables	Dependent Variables	Beta	p value	R^2
Race (O nonwhite, 1 white)	Systolic Blood Pressure	-0.146	0.090	0.077
Gender (0 female, 1 male)		0.100	0.229	
Socioeconomic Status		-0.054	0.532	
Total Vegetables (c)		-0.198	0.018	
Race (O nonwhite, 1 white)	Diastolic Blood Pressure	-0.025	0.776	0.098
Gender (0 female, 1 male)		0.208	0.013	
Socioeconomic Status		-0.145	0.094	
Dietary Fiber (g)		-0.204	0.016	
Race (O nonwhite, 1 white)	High Density Lipoprotein	0.095	0.367	0.103
Gender (0 female, 1 male)		-0.047	0.641	
Socioeconomic Status		0.229	0.032	
Whole Grains (oz eq.)		-0.218	0.033	
Race (O nonwhite, 1 white)	Low Density Lipoprotein	-0.132	0.211	0.100
Gender (0 female, 1 male)		-0.147	0.147	
Socioeconomic Status		0.016	0.877	
Greens and Beans (c)		-0.233	0.023	
Race (O nonwhite, 1 white)	Total Cholesterol	-0.156	0.120	0.187
Gender (0 female, 1 male)		-0.195	0.051	
Socioeconomic Status		0.239	0.017	
Greens and Beans (c)		-0.259	0.007	
Omega-3 Fatty Acids (g)		-0.338	0.003	
Refined Grains (oz eq.)		0.270	0.015	

Race (O nonwhite, 1 white)	Triglycerides	0.253	0.017	0.122
Gender (0 female, 1 male)		-0.103	0.302	
Socioeconomic Status		0.133	0.205	
Dietary Fiber (g)		-0.202	0.049	

Note Race, Gender, and SES were forced entry to use as controls.

Table 12. Relationships of Healthy Eating Index-2010 (HEI) and Body Mass Index (BMI) or Waist Circumference (WC) with Systolic Blood Pressure (SBP), Diastolic Blood Pressure (DBP), High Density Lipoprotein (HDL), Low Density Lipoprotein (LDL), Total Cholesterol (TC), and Triglycerides (TG)

Independent Variable(s)	Dependent Variable	Beta	p value	R^2	Sig. Model	R^2 change	Sig. R^2 change
BMI	SBP	0.418	0.000	0.175	0.000	0.175	0.000
BMI	SBP	0.427	0.000	0.178	0.000	0.003	0.496
HEI		0.053	0.496				
BMI	SBP	0.481	0.000	0.232	0.000	0.054	0.002
HEI		0.095	0.246				
BMI x HEI		0.232	0.002				
BMI	SBP	0.484	0.000	0.237	0.000	0.005	0.338
HEI		0.095	0.214				
BMI x HEI		0.232	0.003				
Gender		0.072	0.338				
BMI	SBP	0.481	0.000	0.237	0.000	0.000	0.900
HEI		0.096	0.214				
BMI x HEI		0.233	0.003				
Gender		0.072	0.340				
Race		0.010	0.900				

BMI	SBP	0.479	0.000	0.238	0.000	0.001	0.691
HEI		0.100	0.201				
BMI x HEI		0.229	0.004				
Gender		0.078	0.313				
Race		0.002	0.976				
SES		-0.032	0.691				

Independent Variable(s)	Dependent Variable	Beta	p value	R^2	Sig. Model	R^2 change	Sig. R^2 change
BMI	DBP	0.343	0.000	0.117	0.000	0.117	0.000
BMI	DBP	0.326	0.000	0.116	0.000	0.011	0.182
HEI		-0.107	0.182				
BMI	DBP	0.353	0.000	0.125	0.000	0.015	0.127
HEI		-0.089	0.273				
BMI x HEI		0.124	0.127				
BMI	DBP	0.360	0.000	0.143	0.000	0.024	0.049
HEI		-0.074	0.355				
BMI x HEI		0.108	0.181				
Gender		0.156	0.049				
BMI	DBP	0.372	0.000	0.138	0.000	0.002	0.584
HEI		-0.078	0.333				
BMI x HEI		0.106	0.193				
Gender		0.156	0.050				
Race		-0.045	0.584				
BMI	DBP	0.362	0.000	0.145	0.000	0.013	0.149
HEI		-0.064	0.430				

BMI x HEI		0.091	0.263				
Gender		0.178	0.027				
Race		-0.073	0.386				
SES		-0.121	0.149				

Independent Variable(s)	Dependent Variable	Beta	p value	R^2	Sig. Model	R^2 change	Sig. R^2 change
BMI	HDL	0.022	0.830	0.000	0.830	0.000	0.830
BMI	HDL	0.017	0.873	0.002	0.929	0.001	0.751
HEI		-0.033	0.751				
BMI	HDL	0.028	0.797	0.004	0.949	0.002	0.647
HEI		-0.026	0.807				
BMI x HEI		0.049	0.647				
BMI	HDL	0.026	0.807	0.005	0.979	0.001	0.767
HEI		-0.029	0.788				
BMI x HEI		0.052	0.629				
Gender		-0.031	0.767				
BMI	HDL	0.064	0.564	0.025	0.806	0.020	0.178
HEI		-0.042	0.696				
BMI x HEI		0.044	0.684				
Gender		-0.030	0.772				
Race		-0.147	0.178				
BMI	HDL	0.084	0.442	0.076	0.300	0.051	0.028
HEI		-0.071	0.504				
BMI x HEI		0.073	0.494				
Gender		-0.075	0.476				

Race		-0.091	0.404				
SES		0.242	0.028				

Independent Variable(s)	Dependent Variable	Beta	p value	R^2	Sig. Model	R^2 change	Sig. R^2 change
BMI	LDL	-0.009	0.930	0.000	0.930	0.000	0.930
BMI HEI	LDL	-0.039 -0.185	0.707 0.075	0.033	0.202	0.033	0.075
BMI HEI BMI x HEI	LDL	-0.035 -0.183 0.018	0.743 0.084 0.867	0.034	0.360	0.000	0.867
BMI HEI BMI x HEI Gender	LDL	-0.042 -0.197 0.035 -0.163	0.692 0.061 0.743 0.114	0.060	0.220	0.026	0.114
BMI HEI BMI x HEI Gender Race	LDL	-0.088 -0.182 0.045 -0.164 0.178	0.418 0.082 0.669 0.109 0.094	0.088	0.128	0.029	0.094
BMI HEI BMI x HEI Gender Race SES	LDL	-0.085 -0.185 0.048 -0.169 0.184 0.027	0.434 0.081 0.651 0.107 0.093 0.800	0.089	0.198	0.001	0.800

Independent Variable(s)	Dependent Variable	Beta	p value	R^2	Sig. Model	R^2 change	Sig. R^2 change
BMI	TC	-0.176	0.085	0.031	0.085	0.031	0.085
BMI HEI	TC	-0.219 -0.272	0.029 0.007	0.103	0.006	0.072	0.007
BMI HEI BMI x HEI	TC	-0.203 -0.261 0.073	0.049 0.011 0.471	0.108	0.014	0.005	0.471
BMI HEI BMI x HEI Gender	TC	-0.212 -0.281 0.096 -0.217	0.036 0.005 0.339 0.028	.0154	0.004	0.046	0.028
BMI HEI BMI x HEI Gender Race	TC	-0.232 -0.274 0.100 -0.217 0.076	0.027 0.007 0.320 0.028 0.452	0.159	0.007	0.005	0.452
BMI HEI BMI x HEI Gender Race SES	TC	-0.213 -0.301 0.127 -0.258 0.128 0.224	0.038 0.003 0.202 0.009 0.210 0.029	0.203	0.002	0.044	0.029

Independent Variable(s)	Dependent Variable	Beta	p value	R^2	Sig. Model	R^2 change	Sig. R^2 change
BMI	TG	-0.040	0.699	0.002	0.699	0.002	0.699
BMI HEI	TG	-0.044 -0.026	0.674 0.801	0.002	0.899	0.001	0.801
BMI HEI BMI x HEI	TG	0.005 0.006 0.220	0.959 0.950 0.038	0.048	0.205	0.046	0.038
BMI HEI BMI x HEI Gender	TG	0.000 -0.005 0.233 -0.126	0.999 0.962 0.028 0.222	0.063	0.194	0.015	0.22
BMI HEI BMI x HEI Gender Race	TG	0.047 -0.021 0.223 -0.125 -0.181	0.665 0.838 0.034 0.220 0.088	0.093	0.108	0.030	0.088
BMI HEI BMI x HEI Gender Race SES	TG	0.062 -0.044 0.246 -0.159 -0.137 0.189	0.560 0.672 0.020 0.121 0.200 0.078	0.124	0.058	0.031	0.078

Independent Variable(s)	Dependent Variable	Beta	p value	R^2	Sig. Model	R^2 change	Sig. R^2 change
WC	SBP	0.412	0.000	0.170	0.000	0.170	0.000
WC HEI	SBP	0.412 0.006	0.000 0.939	0.170	0.000	0.000	0.939
WC HEI WC x HEI	SBP	0.402 0.035 0.115	0.000 0.674 0.163	0.182	0.000	0.012	0.163
WC HEI WC x HEI Gender	SBP	0.403 0.034 0.115 -0.002	0.000 0.678 0.165 0.978	0.182	0.000	0.000	0.978
WC HEI WC x HEI Gender Race	SBP	0.393 0.040 0.121 0.000 0.039	0.000 0.631 0.151 0.999 0.644	0.183	0.000	0.001	0.644
WC HEI WC x HEI Gender Race SES	SBP	0.391 0.44 0.117 0.006 0.031 -0.033	0.000 0.604 0.170 0.939 0.722 0.700	0.184	0.000	0.001	0.700

Independent Variable(s)	Dependent Variable	Beta	p value	R^2	Sig. Model	R^2 change	Sig. R^2 change
WC	DBP	0.342	0.000	0.117	0.000	0.117	0.000
WC HEI	DBP	0.334 -0.142	0.000 0.083	0.137	0.000	0.020	0.083
WC HEI WC x HEI	DBP	0.330 -0.129 0.053	0.000 0.128 0.534	0.139	0.000	0.003	0.534
WC HEI WC x HEI Gender	DBP	0.314 -0.121 0.054 0.091	0.000 0.156 0.520 0.277	0.147	0.000	0.008	0.277
WC HEI WC x HEI Gender Race	DBP	0.320 -0.124 0.051 0.089 -0.023	0.000 0.150 0.552 0.287 0.789	0.148	0.001	0.000	0.789
WC HEI WC x HEI Gender Race SES	DBP	0.311 -0.111 0.036 0.111 -0.051 -0.119	0.000 0.199 0.677 0.191 0.560 0.177	0.160	0.001	0.012	0.177

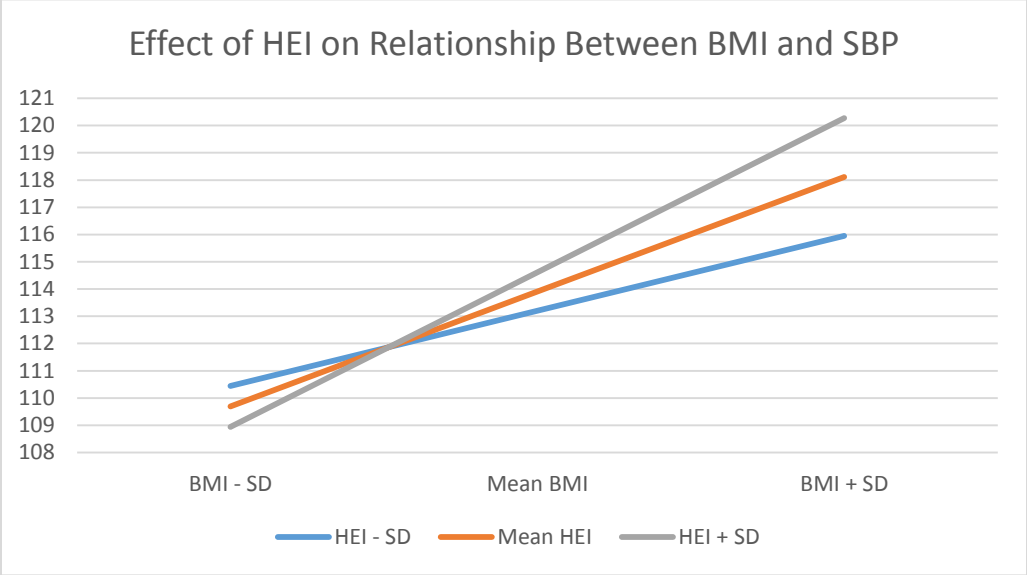
Independent Variable(s)	Dependent Variable	Beta	p value	R^2	Sig. Model	R^2 change	Sig. R^2 change
WC	HDL	0.012	0.911	0.000	0.911	0.000	0.911
WC HEI	HDL	0.010 -0.035	0.925 0.736	0.001	0.939	0.001	0.736
WC HEI WC x HEI	HDL	0.001 -0.011 0.101	0.990 0.922 0.356	0.011	0.804	0.009	0.356
WC HEI WC x HEI Gender	HDL	0.006 -0.013 0.100 -0.028	0.955 0.905 0.361 0.797	0.012	0.902	0.001	0.797
WC HEI WC x HEI Gender Race	HDL	0.038 -0.034 0.080 -0.036 -0.134	0.729 0.759 0.468 0.740 0.226	0.028	0.770	0.016	0.226
WC HEI WC x HEI Gender Race SES	HDL	0.057 -0.061 0.112 -0.083 -0.074 0.250	0.601 0.573 0.304 0.442 0.504 0.026	0.082	0.267	0.054	0.026

Independent Variable(s)	Dependent Variable	Beta	p value	R^2	Sig. Model	R^2 change	Sig. R^2 change
WC	LDL	-0.094	0.369	0.009	0.369	0.009	0.369
WC HEI	LDL	-0.104 -0.184	0.316 0.076	0.043	0.137	0.034	0.076
WC HEI WC x HEI	LDL	-0.103 -0.186 -0.007	0.324 0.084 0.947	0.043	0.266	0.000	0.947
WC HEI WC x HEI Gender	LDL	-0.078 -0.199 -0.010 -0.143	0.458 0.065 0.926 0.175	0.062	0.215	0.020	0.175
WC HEI WC x HEI Gender Race	LDL	-0.121 -0.172 0.017 -0.132 0.178	0.260 0.111 0.876 0.206 0.097	0.092	0.127	0.029	0.097
WC HEI WC x HEI Gender Race SES	LDL	-0.120 -0.174 0.019 -0.136 0.183 0.019	0.270 0.11 0.860 0.205 0.100 0.865	0.092	0.199	0.000	0.865

Independent Variable(s)	Dependent Variable	Beta	p value	R^2	Sig. Model	R^2 change	Sig. R^2 change
WC	TC	-0.215	0.037	0.046	0.037	0.046	0.037
WC HEI	TC	-0.228 -0.249	0.024 0.014	0.108	0.006	0.062	0.014
WC HEI WC x HEI	TC	-0.235 -0.229 0.082	0.021 0.028 0.426	0.114	0.012	0.006	0.426
WC HEI WC x HEI Gender	TC	-0.208 -0.243 0.079 -0.156	0.041 0.019 0.440 0.124	0.138	0.010	0.023	0.124
WC HEI WC x HEI Gender Race	TC	-0.223 -0.233 0.089 -0.152 0.064	0.034 0.027 0.394 0.136 0.538	0.141	0.018	0.004	0.538
WC HEI WC x HEI Gender Race SES	TC	-0.207 -0.257 0.117 -0.193 0.116 0.220	0.046 0.014 0.257 0.059 0.268 0.037	0.183	0.006	0.042	0.037

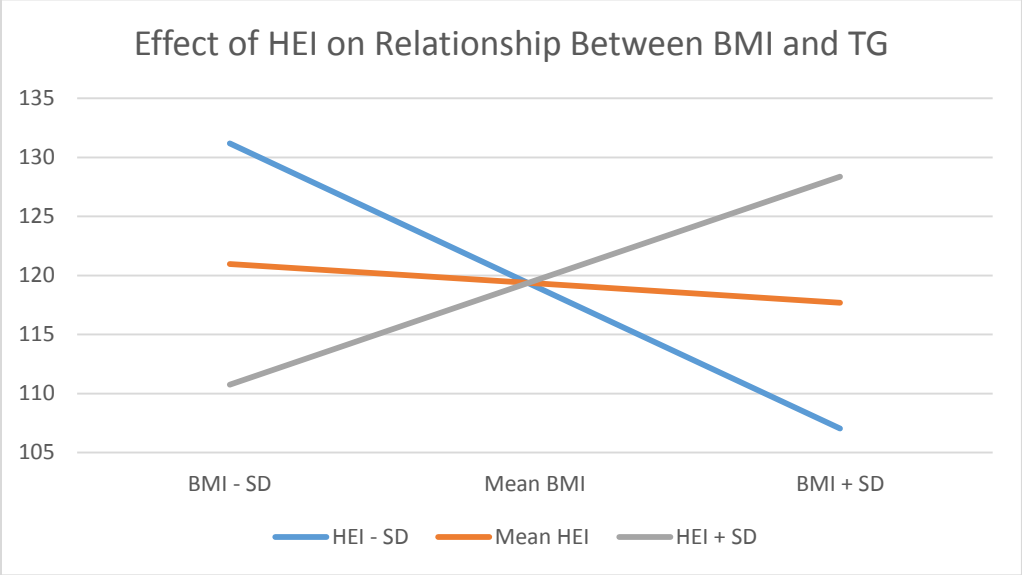
Independent Variable(s)	Dependent Variable	Beta	p value	R^2	Sig. Model	R^2 change	Sig. R^2 change
WC	TG	0.012	0.907	0.000	0.907	0.000	0.907
WC HEI	TG	0.011 -0.019	0.915 0.859	0.000	0.978	0.000	0.859
WC HEI WC x HEI	TG	-0.006 0.032 0.204	0.954 0.768 0.060	0.039	0.305	0.039	0.060
WC HEI WC x HEI Gender	TG	0.011 0.022 0.202 -0.101	0.914 0.836 0.063 0.343	0.049	0.340	0.010	0.343
WC HEI WC x HEI Gender Race	TG	0.056 -0.006 0.175 -0.112 -0.184	0.605 0.953 0.107 0.288 0.088	0.080	0.188	0.031	0.088
WC HEI WC x HEI Gender Race SES	TG	0.070 -0.027 0.199 -0.148 -0.139 0.192	0.512 0.798 0.066 0.164 0.206 0.081	0.112	0.103	0.032	0.081

Figure 1. Effect of Healthy Eating Index-2010 score (HEI) on the Relationship between Body Mass Index (BMI) and Systolic Blood Pressure (SBP)



Y axis represents SBP in mmHg
X axis represents BMI, in kg/m^2, across 1 standard deviation (SD) below mean, mean, and 1 SD above mean
Trend lines represent 1 SD below mean HEI, mean HEI, and 1 SD above mean HEI

Figure 2. Effect of Healthy Eating Index-2010 score (HEI) on the Relationship between Body Mass Index (BMI) and Triglycerides (TG)



Y axis represents TG in mg/dL
X axis represents BMI, in kg/m^2, across 1 standard deviation (SD) below mean, mean, and 1 SD above mean
Trend lines represent 1 SD below mean HEI, mean HEI, and 1 SD above mean HEI

CHAPTER V

EPILOGUE

The results of the studies presented above represent an observation of relationships between diet and adiposity, diet and BP, diet and blood lipids, as well as adiposity and BP, and adiposity and blood lipids among 16 year olds from greater Greensboro, NC. The sample of 163 providing diet information was considered representative of NC at the time of original recruitment, but is no longer representative of the state, consisting of overwhelmingly white and African American participants with minimal population of other racial/ethnic groups. Additionally, this sample has a much greater proportion of females than males. Diet quality, as represented by HEI score, was similarly poor among this sample compared to the HEI score of the same age group for the US. Obesity rate was also similar compared to the US adolescent population. However, rates of hypertension and dyslipidemia were much higher in this sample compared to the US population.

The non-experimental design of this research prohibits establishment of causality in the relationships identified between diet, BMI, WC, BP, and blood lipids. Additionally, the relatively small, predominantly female sample of nearly exclusively white and African American participants prevents extrapolation of results to the greater US population of same age. However, top quality procedures were utilized in collecting diet, anthropometric, and biochemical data, with the exception of a more accurate adiposity

measurement tool. The high quality design of this research, although observational in nature, does allow effective inferences and analysis of relationships identified. In particular, the agreements of key findings presented here with prior research, in both adults and adolescents, adds to a greater understanding of important relationships between diet, body composition, and CVD risk from a young age.

The preponderance of key findings here relate to the interrelationships of obesity, BP, blood lipids, and fruit, vegetable, fiber, and sweetened beverage consumption. Obese participants (by BMI for age) drank more sweetened beverages than non-obese adolescents. Additionally, greater fruit and fiber intakes were related to lower BMI. Similarly, greater fruit and protein intakes were related to lower WC. Obese participants (by both BMI and WC for age) also had higher SBP and DBP compared to non-obese participants, with hypertensive participants having higher BMI and WC than normotensive participants. Increases in both BMI and WC were related to increases in both SBP and DBP. Decreases in SBP were related to increases in vegetable, fiber, and magnesium intakes. Decreases in DBP were related to decreases in fat and increases in total fruit, whole fruit, greens and beans, and fiber intakes. Greens and beans emerge again with relation to blood lipids, as dyslipidemic participants were less likely to meet goal intake of greens and beans than participants with optimal blood lipid profile. Participants with elevated LDL drank more sweetened beverages than those optimal LDL, while those with elevated TC ate fewer omega 3 fatty acids and fiber than those with optimal TC. Decreases in both LDL and TC were related to increases in total vegetable, and greens and beans intakes. Increase in fiber intake was also related to

decreases in both TC and TG. Increases in omega 3 fatty acid consumption, as HEI score were both additionally related to decreases in TC. By contrast, increases in refined grains were related to increases in TC.

Contrary to expectations, a variety of relationships between diet, adiposity, BP, and blood lipids were not observed or were counter to previous findings. Particularly, no relationships were observed between BMI and/or WC with calorie intake, total, or saturated fats, where prior literature has found positive relationships. Inversely, relationships between BMI and/or WC with intake of vegetables and whole grains are not observed here where negative relationships have been previously identified. Additionally, neither BMI nor WC was related to HEI. Unexpectedly, hypertensive participants ate more protein foods, fewer calories, and less trans fat. Similarly contrary to expectations, participants with some form of dyslipidemia (elevated TC, TG, or LDL, or low HDL) had significantly lower WC, and TC was significantly lower in obese participants by BMI for age. Moreover, participants with high TC ate significantly fewer calories, and TC was negatively related to WC. Not previously noted in other research, negative relationships were observed between TC with magnesium and potassium. In conflict with prior research, participants with high TG drank significantly fewer sweetened beverages and ate less added sugar. In addition, increases in whole grain intake were related to decreases in HDL among this sample. However, no relationships were observed between adiposity and LDL, HDL, or TG, nor any relationship between any blood lipids and saturated fats. Lastly, this research did not identify relationships between BP and sodium, potassium, or saturated fatty acids, where increases in sodium and saturated fat intake have previously

been related to increases in BP, with increases in potassium related to decreases in BP. Higher level of aerobic fitness and/or participation in regular athletics may explain a number of these conflicting findings, as athletes may have greater muscle and bone mass, and thus a higher BMI. Moreover, higher aerobic fitness tends to reduce CVD risks, including decrease in BP, TC, and TG, with increase in HDL. Additionally, athletes may rely on higher intake of simple carbohydrates as part of pre, during, and post exercise fueling, as well as take in more total calories than sedentary counterparts. Birth control medication may also play a role in some of these unexpected relationships, as hormonal contraceptives tend to raise TC and HDL. Lastly, the consistently high intakes of sodium and saturated fats, and low intake of potassium across the sample may limit their predictive utility in this study.

The most novel findings of this research were the moderation effects of HEI on the relationships between BMI and CVD risks. In addition to being previously unexplored, the moderation effects observed here revealed an enhancement of CVD risk reduction with better diet quality, but only at a lower BMI. The inverse was observed at higher BMI, which may be a function of athletes eating poorer quality diets or obese participants attempting to improve diet but failing to lose significant weight.

From a public health perspective, the most important finding of this research is the potential role of relatively easily identifiable and large classes of foods as preventive against excess body fatness, HTN, and dyslipidemia. Consistently observed in the results of these chapters are the positive impact of fruits, vegetables, and dietary fiber-rich diets

on BMI, WC, BP, TC, and LDL in terms of reduction of CVD risk. Whole fruits and greens and beans especially emerged as beneficial to BP and blood lipid control. Conversely, a similar but opposite trend was observed for sweetened beverages, which were implicated in obesity by BMI for age and elevations in LDL. Based on these results, continued targeting of incorporation of low calorie density and high fiber plant foods, namely vegetables and fruits, but especially whole fruits, greens, and legumes, is well justified. Simultaneously, the continued push toward limitation of sugar in liquid form also seems well justified. In fact, the results observed here suggest simplification of nutrition messages, at least for the adolescent age group, may be warranted to more greatly focus on the protective aspects of vegetables and fruits and the detrimental effects of sweetened beverages of many varieties. Moreover, these results imply a more simplified and precise focus on increasing vegetable and fruit intake with decrease in sweetened beverage intake may represent a more effective and worthwhile nutrition campaign for adolescents compared to addressing all aspects of whole diet quality.

Traditionally, working with the adolescent population presents a series of difficulties in the research realm. These include the necessity of obtaining both participant consent and parental assent, the common necessity of the parent transporting the participant and inherent increased scheduling conflicts therein, commonly lower reliability of the participant, and development of appropriate incentive structure to recruit and motivate participants. Obtaining physiological data presents additional complexities. Prior to age 18, adolescents commonly have not yet needed blood drawn or any substantial medical testing with any regularity, and fear or trepidation of needles, basic

medical procedures such as BP and pulse measurement, or intimidation from the laboratory setting may all alter results. Commonly referred to as the "white coat syndrome," this false elevation in BP, respiratory rate, and/or heart rate may alter results significantly. More specific to the diet portion of this research, adolescents are typically less in control of their food choices than adults as school lunches or meals prepared by family members are common. As such, adolescents may not be aware of all ingredients in their foods or specific portion sizes.

The research methodology for these studies reflect a number of methods to address these problems. The sample utilized has been tracked for other aspects of a larger research project for many years, providing some familiarity with the research process and high willingness to participate from both parent and participant. Additionally, the majority of data collection occurred during summer and weekends, when schedules are typically more flexible, and allowing easier fasting for the participant prior to blood draw. "White coat syndrome" incidence was likely reduced by the procedures upon arrival to the lab. First, relatively young research assistants, not wearing traditional medical gear or lab coats, greeted and brought the participant into the UNCG exercise physiology lab, which was large and open, a very different environment compared to most medical facilities. Second, consent and assent were obtained as the participant was seated and procedures to come were explained. Third, NHLBI standards were adhered to for BP and pulse measurements, with the participant seated a minimum of five minutes, and with multiple repeated measurements. Fourth, the blood draw was obtained in a comfortable, reclined chair.

For collecting diet information, this research utilized the highest quality method for research, multiple pass diet recall. This method allows recall of increasingly specific diet information and prevents forgotten foods or beverages through its repeated pass design. Additionally, trained researchers with the UNC Nutrition Obesity Research Center made calls to participants, usually their cellular phones, which allowed convenience for the participant and ease of access for the researcher. Furthermore, the compensation structure for the participants incentivized full completion, as a flat rate was provided for the visit to the lab, followed by increasing dollar values for gift cards for completion of each successive diet recall. The primary problem encountered in this research was a significantly delayed timeframe for completion of the diet recalls. Although the target timeline was to complete three diet recalls per participant by two weeks after the visit to the lab, the first completed diet recall was routinely over 6 weeks after the lab visit. This may have allowed significant alterations to diet compared to diet leading up to the lab visit. In addition, this led to delays in delivery of data and timeline for completion of aspects of the research. Otherwise, problems encountered were very minor or essentially unavoidable, such as same day or short notice rescheduling from participants, inability to actually contact the participant for recruitment, or adverse reactions to testing that were previously unknown.

This research has considerable potential for future exploration. In particular, a variety of other key aspects of general health and measures for CVD risk may be explored. Future research should examine aerobic fitness, in the form of VO2max testing, type 2 diabetes and metabolic syndrome risks such as homeostatic model of assessment

of insulin resistance (HOMA-IR) as well as a variety of liver enzymes. Greater exploration into low grade elevations to inflammatory markers should also be considered, such as c-reactive protein (CRP), and a variety of inflammatory cytokines like TNFα, IL-6, and IL-10. More accurate measures of body composition should also be considered for future research, such as underwater weighing, BodPod®, or advanced bioelectrical impedance analysis machines like the Seca mBCA or InBody 770, to determine body fatness.

In an ideal scenario, future research could recruit a larger and more representative sample to conduct specific interventions. Particularly based on these results, a study would be structured with intervention groups counseled and/or provided incentives to increase vegetable and fruit intake with and without decreases in sweetened beverage consumption, along with an intervention group counseled and/or incentivized to decrease sweetened beverage consumption only, and a control group incentivized to avoid dietary alterations compared to baseline. Similar anthropometric and biochemical data could be measured and analyzed at baseline and endpoint, ideally with the additional measures noted above. Greater exploration into the moderation effects of diet quality in relationships to predict CVD risks should be emphasized to understand the most important populations to target for dietary improvement. Additional research may also examine the effects of improvements in diet quality on CVD risks independent of changes in body composition, and the effects of improvements in body composition on CVD risk independent of diet quality improvements. These proposed studies would provide causal insights into various aspects of health and wellness on CVD risk, whether

via body composition alteration, additions to diet, restrictions to diet, and general overall diet quality improvement. Those results, in turn, would allow alteration and possible simplification of public health messaging and campaigns to maximize morbidity risk outcomes.

From a personal and professional perspective, this research has had a profound impact on the researcher himself. Over the course of this research, I learned the overhelmingly complex and laborious nature of larger and longer term research. I was immediately surprised by how large the team needed to be for adequate functionality of the Right Track research progression, let alone the portion of data attributed to my project. I quickly became appreciative of the thoroughness and time required to handle large pools of data, recuit and retain participants, and arrange for continued academic output for literature. On one hand, working with all this data, becoming familiar with the numbers intimately and routinely reviewing, updating, and analyzing this data had led me to genuinely appreciate the objective and independent nature of data analysis and statistics. Additionally, it has led me to strive, often to the point of annoying or impressing my colleagues (depending on the person), to increase efficiency and transparency in all my work. This led me to overhaul note templates at clinics for whom I've worked, update practices for entering and handling survey data at military foodservice facilities, tracking patient flow at other clinics, and calculate appropriate patient work load expectations for RDs at multiple other sites. On another hand, the experience was so engrossing that I became hopeful of a break. In all honesty, I consider my total research, teaching, and general PhD experience to have been wildly valuable in

identifying, interpreting, and utilizing research for my clinical practice, as well as directly applicable to generally increasing the efficiency of my lifestyle, leadership, and management. However, I am not sure a career in research is for me- I find that leadership positions and part time patient contact have been my favorite combination of roles over the course of the past few years. Considering my newest position with the Navy, though, this experience sets me up perfectly. In fact, I need to collect, manage, and interpret data on a weekly basis at my next position, and make leadership decisions based upon the information available. As such, it would seem things have fallen into place, and this experience has changed me a great deal. I consider myself now a more efficient person and more effective leader and clinician. Even if I never return to research, this work changed my life for the better.

REFERENCES

1. Ogden CL, Carroll MD, Kit BK, Flegal KM. PRevalence of obesity and trends in body mass index among us children and adolescents, 1999-2010. JAMA. 2012;307:483–90.

2. Hiza HAB, Guenther PM, Rihane CI. Diet Quality of Children Age 2-17 Years as Measured by the Healthy Eating Index-2010 [Internet]. United States Department of Agriculture Center for Nutrition Policy and Promotion; 2013 Jul. Report No.: 52. Available from: http://www.cnpp.usda.gov/HealthyEatingIndex.htm

3. Fungwe T, Guenther PM, Juan W, Hiza HAB, Lino M. The Quality of Children's Diets in 2003-04 as Measured by the Healthy Eating Index-2005. Center for Nutrition Policy and Promotion: United States Department of Agriculture; 2009 Apr. Report No.: 43.

4. O'Neil CE, Nicklas TA, Zanovec M, Fulgoni VL. Diet quality is positively associated with 100% fruit juice consumption in children and adults in the United States: NHANES 2003-2006. Nutr J. 2011;10:17.

5. Beydoun MA, Powell LM, Chen X, Wang Y. Food Prices Are Associated with Dietary Quality, Fast Food Consumption, and Body Mass Index among U.S. Children and Adolescents. J Nutr. 2011;141:304–11.

6. Mikkilä V, Räsänen L, Raitakari O t., Pietinen P, Viikari J. Consistent dietary patterns identified from childhood to adulthood: The Cardiovascular Risk in Young Finns Study. Br J Nutr. 2005;93:923–31.

7. Centers for Disease Control and Prevention (CDC). Prevalence of abnormal lipid levels among youths --- United States, 1999-2006. MMWR Morb Mortal Wkly Rep. 2010;59:29–33.

8. Berenson GS, Srinivasan SR, Bao W, Newman WP 3rd, Tracy RE, Wattigney WA. Association between multiple cardiovascular risk factors and atherosclerosis in children and young adults. The Bogalusa Heart Study. N Engl J Med. 1998;338:1650–6.

9. Enos WF, Holmes RH, Beyer J. Coronary disease among United States soldiers killed in action in Korea; preliminary report. J Am Med Assoc. 1953;152:1090–3.

10. Strong JP, Malcom GT, McMahan C, et al. Prevalence and extent of atherosclerosis in adolescents and young adults: Implications for prevention from the pathobiological determinants of atherosclerosis in youth study. JAMA. 1999;281:727–35.

11. Holman RL, McGill HC Jr, Strong JP, Geer JC. The natural history of atherosclerosis: the early aortic lesions as seen in New Orleans in the middle of the of the 20th century. Am J Pathol. 1958;34:209–35.

12. Olsen R. Atherogenesis in children: implications for the prevention of athersclerosis. Adv Pediatr. 2000;47:55–78.

13. Celermajer, David S. Endothelial Dysfunction: Does It Matter? Is It Reversible? J Am Coll Cardiol. 1997;30:325–33.

14. Epstein LH, Gordy CC, Raynor HA, Beddome M, Kilanowski CK, Paluch R. Increasing Fruit and Vegetable Intake and Decreasing Fat and Sugar Intake in Families at Risk for Childhood Obesity. Obes Res. 2001;9:171–8.

15. Ludwig DS, Peterson KE, Gortmaker SL. Relation between consumption of sugar-sweetened drinks and childhood obesity: a prospective, observational analysis. The Lancet. 2001;357:505–8.

16. Ludwig DS, Pereira MA, Kroenke CH, et al. DIetary fiber, weight gain, and cardiovascular disease risk factors in young adults. JAMA. 1999;282:1539–46.

17. Sacks FM, Bray GA, Carey VJ, Smith SR, Ryan DH, Anton SD, McManus K, Champagne CM, Bishop LM, Laranjo N, et al. Comparison of Weight-Loss Diets with Different Compositions of Fat, Protein, and Carbohydrates. N Engl J Med. 2009;360:859–73.

18. Pereira MA, Kartashov AI, Ebbeling CB, Van Horn L, Slattery ML, Jacobs Jr DR, Ludwig DS. Fast-food habits, weight gain, and insulin resistance (the CARDIA study): 15-year prospective analysis. The Lancet. 2005;365:36–42.

19. Guenther PM, Kirkpatrick SI, Reedy J, Krebs-Smith SM, Buckman DW, Dodd KW, Casavale KO, Carroll RJ. The Healthy Eating Index-2010 Is a Valid and Reliable Measure of Diet Quality According to the 2010 Dietary Guidelines for Americans. J Nutr. 2014;jn.113.183079.

20. Voulgari C, Tentolouris N, Dilaveris P, Tousoulis D, Katsilambros N, Stefanadis C. Increased Heart Failure Risk in Normal-Weight People With Metabolic Syndrome Compared With Metabolically Healthy Obese Individuals. J Am Coll Cardiol. 2011;58:1343–50.

21. Janssen I. Heart disease risk among metabolically healthy obese men and metabolically unhealthy lean men. Can Med Assoc J. 2005;172:1315–6.

22. Blüher M. The distinction of metabolically “healthy” from “unhealthy” obese individuals: Curr Opin Lipidol. 2010;21:38–43.

23. Shah RV, Abbasi SA, Neilan TG, Hulten E, Coelho-Filho O, Hoppin A, Levitsky L, Ferranti S de, Rhodes ET, Traum A, et al. Myocardial Tissue Remodeling in Adolescent Obesity. J Am Heart Assoc. 2013;2:e000279.

24. Balakrishnan PL. Identification of Obesity and Cardiovascular Risk Factors in Childhood and Adolescence. Pediatr Clin North Am. 2014;61:153–71.

25. McGill HC, McMahan CA, Gidding SS. Preventing Heart Disease in the 21st Century Implications of the Pathobiological Determinants of Atherosclerosis in Youth (PDAY) Study. Circulation. 2008;117:1216–27.

26. Friedemann C, Heneghan C, Mahtani K, Thompson M, Perera R, Ward AM. Cardiovascular disease risk in healthy children and its association with body mass index: systematic review and meta-analysis. BMJ. 2012;345:e4759.

27. Baker JL, Olsen LW, Sørensen TIA. Childhood body-mass index and the risk of coronary heart disease in adulthood. N Engl J Med. 2007;357:2329–37.

28. De Moraes ACF, Fadoni RP, Ricardi LM, Souza TC, Rosaneli CF, Nakashima ATA, Falcão MC. Prevalence of abdominal obesity in adolescents: a systematic review. Obes Rev. 2011;12:69–77.

29. Cook S, Weitzman M, Auinger P, Nguyen M, Dietz WH. Prevalence of a metabolic syndrome phenotype in adolescents: findings from the third National Health and Nutrition Examination Survey, 1988-1994. Arch Pediatr Adolesc Med. 2003;157:821–7.

30. Li S, Chen W, Srinivasan SR, Bond MG, Tang R, Urbina EM, Berenson GS. Childhood cardiovascular risk factors and carotid vascular changes in adulthood: the Bogalusa Heart Study. JAMA J Am Med Assoc. 2003;290:2271–6.

31. Slyper AH, Rosenberg H, Kabra A, Weiss MJ, Blech B, Gensler S, Matsumura M. Early atherogenesis and visceral fat in obese adolescents. Int J Obes 2005. 2014;

32. Keefer DJ, Caputo JL, Tseh W. Waist-to-Height Ratio and Body Mass Index as Indicators of Cardiovascular Risk in Youth. J Sch Health. 2013;83:805–9.

33. Burgos MS, Burgos LT, Camargo MD, Franke SIR, Prá D, Silva AMV da, Borges TS, Todendi PF, Reckziegel MB, Reuter CP. Relationship between anthropometric measures and cardiovascular risk factors in children and adolescents. Arq Bras Cardiol. 2013;101:288–96.

34. Reuter CP, Burgos LT, Camargo MD, Possuelo LG, Reckziegel MB, Reuter EM, Meinhardt FP, Burgos MS, Reuter CP, Burgos LT, et al. Prevalence of obesity and cardiovascular risk among children and adolescents in the municipality of Santa Cruz do Sul, Rio Grande do Sul. Sao Paulo Med J. 2013;131:323–30.

35. Expert Panel on Integrated Guidelines for Cardiovascular Health and Risk Reduction in Children and Adolescents, National Heart, Lung, and Blood Institute. Expert panel on integrated guidelines for cardiovascular health and risk reduction in children and adolescents: summary report. Pediatrics. 2011;128 Suppl 5:S213–56.

36. Magnussen CG, Niinikoski H, Juonala M, Kivimäki M, Rönnemaa T, Viikari JSA, Simell O, Raitakari OT. When and how to start prevention of atherosclerosis? Lessons from the Cardiovascular Risk in the Young Finns Study and the Special Turku Coronary Risk Factor Intervention Project. Pediatr Nephrol Berl Ger. 2012;27:1441–52.

37. Kavey R-EW, Daniels SR, Lauer RM, Atkins DL, Hayman LL, Taubert K, American Heart Association. American Heart Association guidelines for primary prevention of atherosclerotic cardiovascular disease beginning in childhood. J Pediatr. 2003;142:368–72.

38. May AL, Kuklina EV, Yoon PW. Prevalence of Cardiovascular Disease Risk Factors Among US Adolescents, 1999−2008. Pediatrics. 2012;129:1035–41.

39. Al-Attas OS, Al-Daghri NM, Alokail MS, Alkharfy KM, Draz H, Yakout S, Sabico S, Chrousos G. Association of body mass index, sagittal abdominal diameter and waist-hip ratio with cardiometabolic risk factors and adipocytokines in Arab children and adolescents. BMC Pediatr. 2012;12:119.

40. Pereira PF, Serrano HMS, Carvalho GQ, Lamounier JA, Peluzio M do CG, Franceschini S do CC, Priore SE. Body fat location and cardiovascular disease risk factors in overweight female adolescents and eutrophic female adolescents with a high percentage of body fat. Cardiol Young. 2012;22:162–9.

41. Namburi RP, Ponnala AR, Karthik TS, Rani PR, Maheshwari R. A study on metabolic variables and its association with high sensitive C-reactive protein in obese children and adolescents. Indian J Endocrinol Metab. 2013;17:S360–2.

42. Ryder JR, Vega-López S, Djedjos CS, Shaibi GQ. Abdominal adiposity, insulin resistance, and oxidized low-density lipoproteins in Latino adolescents. Diabetol Metab Syndr. 2013;5:72.

43. Posadas-Sánchez R, Posadas-Romero C, Zamora-González J, Mendoza-Pérez E, Cardoso-Saldaña G, Yamamoto-Kimura L. Lipid and lipoprotein profiles and prevalence of dyslipidemia in Mexican adolescents. Metab - Clin Exp. 2007;56:1666–72.

44. Teixeira PJ, Sardinha LB, Going SB, Lohman TG. Total and Regional Fat and Serum Cardiovascular Disease Risk Factors in Lean and Obese Children and Adolescents. Obes Res. 2001;9:432–42.

45. Lee S, Bacha F, Arslanian SA. Waist circumference, blood pressure, and lipid components of the metabolic syndrome. J Pediatr. 2006;149:809–16.

46. Jago R, Harrell JS, McMurray RG, Edelstein S, El Ghormli L, Bassin S. Prevalence of abnormal lipid and blood pressure values among an ethnically diverse population of eighth-grade adolescents and screening implications. Pediatrics. 2006;117:2065–73.

47. Plourde G. Impact of obesity on glucose and lipid profiles in adolescents at different age groups in relation to adulthood. BMC Fam Pract. 2002;3:18.

48. Spinneker A, Egert S, González-Gross M, Breidenassel C, Albers U, Stoffel-Wagner B, Huybrechts I, Manios Y, Venneria E, Molnar D, et al. Lipid, lipoprotein and apolipoprotein profiles in European adolescents and its associations with gender, biological maturity and body fat—The HELENA Study. Eur J Clin Nutr. 2012;66:727–35.

49. Thompson DR, Obarzanek E, Franko DL, Barton BA, Morrison J, Biro FM, Daniels SR, Striegel-Moore RH. Childhood Overweight and Cardiovascular Disease Risk Factors: The National Heart, Lung, and Blood Institute Growth and Health Study. J Pediatr. 2007;150:18–25.

50. Tresaco B, Moreno LA, Ruiz JR, Ortega FB, Bueno G, González-Gross M, Wärnberg J, Gutiérrez A, García-Fuentes M, Marcos A, et al. Truncal and Abdominal Fat as Determinants of High Triglycerides and Low HDL-cholesterol in Adolescents. Obesity. 2009;17:1086–91.

51. Plachta-Danielzik S, Landsberg B, Johannsen M, Lange D, Müller MJ. Association of different obesity indices with blood pressure and blood lipids in children and adolescents. Br J Nutr. 2008;100:208–18.

52. Ambrosini GL, Huang R-C, Mori TA, Hands BP, O'Sullivan TA, de Klerk NH, Beilin LJ, Oddy WH. Dietary patterns and markers for the metabolic syndrome in Australian adolescents. Nutr Metab Cardiovasc Dis. 2010;20:274–83.

53. Dai S, Fulton JE, Harrist RB, Grunbaum JA, Steffen LM, Labarthe DR. Blood lipids in children: age-related patterns and association with body-fat indices: Project HeartBeat! Am J Prev Med. 2009;37:S56–64.

54. Tracy RE, Newman WP 3rd, Wattigney WA, Srinivasan SR, Strong JP, Berenson GS. Histologic features of atherosclerosis and hypertension from autopsies of young individuals in a defined geographic population: the Bogalusa Heart Study. Atherosclerosis. 1995;116:163–79.

55. Chamontin B, Amar J, Barthe P, Salvador M. Blood pressure measurements and left ventricular mass in young adults with arterial hypertension screened at high school check-up. J Hum Hypertens. 1994;8:357–61.

56. McNiece KL, Gupta-Malhotra M, Samuels J, Bell C, Garcia K, Poffenbarger T, Sorof JM, Portman RJ, National High Blood Pressure Education Program Working Group. Left ventricular hypertrophy in hypertensive adolescents: analysis of risk by 2004 National High Blood Pressure Education Program Working Group staging criteria. Hypertension. 2007;50:392–5.

57. Drukteinis JS, Roman MJ, Fabsitz RR, Lee ET, Best LG, Russell M, Devereux RB. Cardiac and systemic hemodynamic characteristics of hypertension and prehypertension in adolescents and young adults: the Strong Heart Study. Circulation. 2007;115:221–7.

58. Anyaegbu EI, Dharnidharka VR. Hypertension in the Teenager. Pediatr Clin North Am. 2014;61:131–51.

59. Bao W, Threefoot SA, Srinivasan SR, Berenson GS. Essential hypertension predicted by tracking of elevated blood pressure from childhood to adulthood: the Bogalusa Heart Study. Am J Hypertens. 1995;8:657–65.

60. Beckett LA, Rosner B, Roche AF, Guo S. Serial changes in blood pressure from adolescence into adulthood. Am J Epidemiol. 1992;135:1166–77.

61. Ostchega Y, Carroll M, Prineas RJ, McDowell MA, Louis T, Tilert T. Trends of Elevated Blood Pressure Among Children and Adolescents: Data From the National Health and Nutrition Examination Survey 1988–2006. Am J Hypertens. 2009;22:59–67.

62. McNiece KL, Poffenbarger TS, Turner JL, Franco KD, Sorof JM, Portman RJ. Prevalence of Hypertension and Pre-Hypertension among Adolescents. J Pediatr. 2007;150:640–4.e1.

63. Acosta AA, Samuels JA, Portman RJ, Redwine KM. Prevalence of Persistent Prehypertension in Adolescents. J Pediatr. 2012;160:757–61.

64. Sorof JM, Lai D, Turner J, Poffenbarger T, Portman RJ. Overweight, Ethnicity, and the Prevalence of Hypertension in School-Aged Children. Pediatrics. 2004;113:475–82.

65. Katona É, Zrínyi M, Lengyel S, Komonyi É, Paragh G, Zatik J, Nagy G, Fülesdi B, Páll D. The prevalence of adolescent hypertension in Hungary – The Debrecen Hypertension Study. Blood Press. 2011;20:134–9.

66. Cao Z, Zhu L, Zhang T, Wu L, Wang Y. Blood Pressure and Obesity Among Adolescents: A School-Based Population Study in China. Am J Hypertens. 2012;25:576–82.

67. McCrindle BW, Manlhiot C, Millar K, Gibson D, Stearne K, Kilty H, Prentice D, Wong H, Chahal N, Dobbin SW. Population Trends Toward Increasing Cardiovascular Risk Factors in Canadian Adolescents. J Pediatr. 2010;157:837–43.

68. Magliano ES, Guedes LG, Coutinho ESF, Bloch KV. Prevalence of arterial hypertension among Brazilian adolescents: systematic review and meta-analysis. BMC Public Health. 2013;13:833.

69. Silva D, Matos A, Magalhães T, Martins V, Ricardo L, Almeida H. Prevalência de hipertensão arterial em adolescentes portugueses da cidade de Lisboa. Rev Port Cardiol. 2012;31:789–94.

70. Lichtenstein AH, Appel LJ, Brands M, Carnethon M, Daniels S, Franch HA, Franklin B, Kris-Etherton P, Harris WS, Howard B, et al. Diet and Lifestyle Recommendations Revision 2006 A Scientific Statement From the American Heart Association Nutrition Committee. Circulation. 2006;114:82–96.

71. Hill JO, Melanson EL, Wyatt HT. Dietary Fat Intake and Regulation of Energy Balance: Implications for Obesity. J Nutr. 2000;130:284S – 288S.

72. Webber J. Energy balance in obesity. Proc Nutr Soc. 2003;62:539–43.

73. Howarth NC, Huang TT-K, Roberts SB, McCrory MA. Dietary Fiber and Fat Are Associated with Excess Weight in Young and Middle-Aged US Adults. J Am Diet Assoc. 2005;105:1365–72.

74. Field AE, Willett WC, Lissner L, Colditz GA. Dietary Fat and Weight Gain Among Women in the Nurses' Health Study. Obesity. 2007;15:967–76.

75. Millen BE, Pencina MJ, Kimokoti RW, Zhu L, Meigs JB, Ordovas JM, D'Agostino RB. Nutritional risk and the metabolic syndrome in women: opportunities for preventive intervention from the Framingham Nutrition Study. Am J Clin Nutr. 2006;84:434–41.

76. Du H, van der A DL, Ginder V, Jebb SA, Forouhi NG, Wareham NJ, Halkjær J, Tjønneland A, Overvad K, Jakobsen MU, et al. Dietary Energy Density in Relation to Subsequent Changes of Weight and Waist Circumference in European Men and Women. Stanojevic S, editor. PLoS ONE. 2009;4:e5339.

77. Slavin JL. Dietary fiber and body weight. Nutrition. 2005;21:411–8.

78. Lairon D, Arnault N, Bertrais S, Planells R, Clero E, Hercberg S, Boutron-Ruault M-C. Dietary fiber intake and risk factors for cardiovascular disease in French adults. Am J Clin Nutr. 2005;82:1185–94.

79. Wirfält E, Hedblad B, Gullberg B, Mattisson I, Andrén C, Rosander U, Janzon L, Berglund G. Food Patterns and Components of the Metabolic Syndrome in Men and Women: A Cross-sectional Study within the Malmö Diet and Cancer Cohort. Am J Epidemiol. 2001;154:1150–9.

80. DiBello JR, McGarvey ST, Kraft P, Goldberg R, Campos H, Quested C, Laumoli TS, Baylin A. Dietary Patterns Are Associated with Metabolic Syndrome in Adult Samoans. J Nutr. 2009;139:1933–43.

81. Newby PK, Muller D, Hallfrisch J, Qiao N, Andres R, Tucker KL. Dietary patterns and changes in body mass index and waist circumference in adults. Am J Clin Nutr. 2003;77:1417–25.

82. Field AE, Gillman MW, Rosner B, Rockett HR, Colditz GA. Association between fruit and vegetable intake and change in body mass index among a large sample of children and adolescents in the United States. Int J Obes. 2003;27:821–6.

83. He K, Hu FB, Colditz GA, Manson JE, Willett WC, Liu S. Changes in intake of fruits and vegetables in relation to risk of obesity and weight gain among middle-aged women. Int J Obes. 2004;28:1569–74.

84. Lin B-H, Morrison RM. Higher Fruit Consumption Linked With Lower Body Mass Index. FoodReview. 2002;25:28–32.

85. Rolls BJ, Ello-Martin JA, Tohill BC. What Can Intervention Studies Tell Us about the Relationship between Fruit and Vegetable Consumption and Weight Management? Nutr Rev. 2004;62:1–17.

86. Tohill BC, Seymour J, Serdula M, Kettel-Khan L, Rolls BJ. What Epidemiologic Studies Tell Us about the Relationship between Fruit and Vegetable Consumption and Body Weight. Nutr Rev. 2004;62:365–74.

87. Collison KS, Zaidi MZ, Subhani SN, Al-Rubeaan K, Shoukri M, Al-Mohanna FA. Sugar-sweetened carbonated beverage consumption correlates with BMI, waist circumference, and poor dietary choices in school children. BMC Public Health. 2010;10:234.

88. Kent L, Worsley A. Trends in BMI, diet and lifestyle between 1976 and 2005 in North Sydney. Fac Health Behav Sci - Pap Arch. 2009;453–61.

89. Te Morenga L, Mallard S, Mann J. Dietary sugars and body weight: systematic review and meta-analyses of randomised controlled trials and cohort studies. BMJ. 2012;346:e7492–e7492.

90. Schröder H, Fïto M, Covas MI. Association of fast food consumption with energy intake, diet quality, body mass index and the risk of obesity in a representative Mediterranean population. Br J Nutr. 2007;98:1274–80.

91. Sacks FM, Svetkey LP, Vollmer WM, Appel LJ, Bray GA, Harsha D, Obarzanek E, Conlin PR, Miller ER, Simons-Morton DG, et al. Effects on Blood Pressure of Reduced Dietary Sodium and the Dietary Approaches to Stop Hypertension (DASH) Diet. N Engl J Med. 2001;344:3–10.

92. Blumenthal JA, Babyak MA, Hinderliter A, et al. Effects of the dash diet alone and in combination with exercise and weight loss on blood pressure and cardiovascular biomarkers in men and women with high blood pressure: The encore study. Arch Intern Med. 2010;170:126–35.

93. Vollmer WM, Sacks FM, Ard J, Appel LJ, Bray GA, Simons-Morton DG, Conlin PR, Svetkey LP, Erlinger TP, Moore TJ, et al. Effects of Diet and Sodium Intake on Blood Pressure: Subgroup Analysis of the DASH-Sodium Trial. Ann Intern Med. 2001;135:1019–28.

94. Hermansen K. Diet, blood pressure and hypertension. Br J Nutr. 2000;83:S113–9.

95. Pimenta E, Gaddam KK, Oparil S, Aban I, Husain S, Dell'Italia LJ, Calhoun DA. Effects of Dietary Sodium Reduction on Blood Pressure in Subjects With Resistant Hypertension Results From a Randomized Trial. Hypertension. 2009;54:475–81.

96. Graudal NA, Hubeck-Graudal T, Jürgens G. Effects of low-sodium diet vs. high-sodium diet on blood pressure, renin, aldosterone, catecholamines, cholesterol, and triglyceride (Cochrane Review). Am J Hypertens. 2012;25:1–15.

97. Mu JJ, Liu ZQ, Liu WM, Liang YM, Yang DY, Zhu DJ, Wang ZX. Reduction of blood pressure with calcium and potassium supplementation in children with salt sensitivity: a 2-year double-blinded placebo-controlled trial. J Hum Hypertens. 2005;19:479–83.

98. He FJ, Li J, Macgregor GA. Effect of longer term modest salt reduction on blood pressure: Cochrane systematic review and meta-analysis of randomised trials. BMJ. 2013;346:f1325.

99. Haddy FJ, Vanhoutte PM, Feletou M. Role of potassium in regulating blood flow and blood pressure. Am J Physiol - Regul Integr Comp Physiol. 2006;290:R546–52.

100. Geleijnse JM, Kok FJ, Grobbee DE. Blood pressure response to changes in sodium and potassium intake: a metaregression analysis of randomised trials. J Hum Hypertens. 2003;17:471–80.

101. Van Mierlo LAJ, Arends LR, Streppel MT, Zeegers MPA, Kok FJ, Grobbee DE, Geleijnse JM. Blood pressure response to calcium supplementation: a meta-analysis of randomized controlled trials. J Hum Hypertens. 2006;20:571–80.

102. Griffith LE, Guyatt GH, Cook RJ, Bucher HC, Cook DJ. The influence of dietary and nondietary calcium supplementation on blood pressure: An updated metaanalysis of randomized controlled trials. Am J Hypertens. 1999;12:84–92.

103. Jee SH, Miller ER, Guallar E, Singh VK, Appel LJ, Klag MJ. The effect of magnesium supplementation on blood pressure: a meta-analysis of randomized clinical trials. Am J Hypertens. 2002;15:691–6.

104. Geleijnse JM, Giltay EJ, Grobbee DE, Donders ART, Kok FJ. Blood pressure response to fish oil supplementation: metaregression analysis of randomized trials. J Hypertens. 2002;20:1493–9.

105. Cicero AFG, Ertek S, Borghi C. Omega-3 Polyunsaturated Fatty Acids: Their Potential Role in Blood Pressure Prevention and Management. Curr Vasc Pharmacol. 2009;7:330–7.

106. Ueshima H, Stamler J, Elliott P, Chan Q, Brown IJ, Carnethon MR, Daviglus ML, He K, Moag-Stahlberg A, Rodriguez BL, et al. Food Omega-3 Fatty Acid Intake of Individuals (Total, Linolenic Acid, Long-Chain) and Their Blood Pressure INTERMAP Study. Hypertension. 2007;50:313–9.

107. Liu JC, Conklin SM, Manuck SB, Yao JK, Muldoon MF. Long-Chain Omega-3 Fatty Acids and Blood Pressure. Am J Hypertens. 2011;24:1121–6.

108. Lungerhausen Y, Abbey M, Nestel P, Howe P. Reduction of blood pressure and plasma triglycerides by omega-3 fatty acids in treated hypertensives. J Hypertens. 1994;12:1041–5.

109. Washi SA, Ageib MB. Poor diet quality and food habits are related to impaired nutritional status in 13- to 18-year-old adolescents in Jeddah. Nutr Res. 2010;30:527–34.

110. Harika RK, Cosgrove MC, Osendarp SJM, Verhoef P, Zock PL. Fatty acid intakes of children and adolescents are not in line with the dietary intake recommendations for future cardiovascular health: a systematic review of dietary intake data from thirty countries. Br J Nutr. 2011;106:307–16.

111. Sanders TA, Oakley FR, Miller GJ, Mitropoulos KA, Crook D, Oliver MF. Influence of n-6 versus n-3 polyunsaturated fatty acids in diets low in saturated fatty acids on plasma lipoproteins and hemostatic factors. Arterioscler Thromb Vasc Biol. 1997;17:3449–60.

112. Welsh JA, Sharma A, Cunningham SA, Vos MB. Consumption of Added Sugars and Indicators of Cardiovascular Disease Risk Among US Adolescents. Circulation. 2011;123:249–57.

113. Bradlee ML, Singer MR, Qureshi MM, Moore LL. Food group intake and central obesity among children and adolescents in the Third National Health and Nutrition Examination Survey (NHANES III). Public Health Nutr. 2010;13:797–805.

114. Bel-Serrat S, Mouratidou T, Jiménez-Pavón D, Huybrechts I, Cuenca-García M, Mistura L, Gottrand F, González-Gross M, Dallongeville J, Kafatos A, et al. Is dairy consumption associated with low cardiovascular disease risk in European adolescents? Results from the HELENA Study. Pediatr Obes. 2014;9:401–10.

115. Mohseni-Takalloo S, Mirmiran P, Hosseini-Esfahani F, Mehrabi Y, Azizi F. Metabolic Syndrome and its Association with Healthy Eating Index-2005 in Adolescents: Tehran Lipid and Glucose Study. J Food Nutr Res. 2014;2:155–61.

116. Food Patterns Equivalents Intakes from Food: Mean Amounts Consumed per Individual, by Gender and Age. [Internet]. U.S. Department of Agriculture, Agricultural Research Service; 2013. Available from: www.ars.usda.gov/ba/bhnrc/fsrg

117. Nutrient Intakes from Food: Mean Amounts Consumed per Individual, by Gender and Age [Internet]. U.S. Department of Agriculture, Agricultural Research Service; 2012. Available from: www.ars.usda.gov/ba/bhnrc/fsrg

118. Otten JJ, Pitzi H, Jennifer, Meyers LD. DRI, Dietary Reference Intakes: The Essential Guide to Nutrient Requirements. National Academies Press; 2006. 1345 p.

119. Striegel-Moore RH, Thompson D, Affenito SG, Franko DL, Obarzanek E, Barton BA, Schreiber GB, Daniels SR, Schmidt M, Crawford PB. Correlates of beverage intake in adolescent girls: The National Heart, Lung, and Blood Institute Growth and Health Study. J Pediatr. 2006;148:183–7.

120. Libuda L, Alexy U, Sichert-Hellert W, Stehle P, Karaolis-Danckert N, Buyken AE, Kersting M. Pattern of beverage consumption and long-term association with body-weight status in German adolescents – results from the DONALD study. Br J Nutr. 2008;99:1370–9.

121. Francis DK, Van den Broeck J, Younger N, McFarlane S, Rudder K, Gordon-Strachan G, Grant A, Johnson A, Tulloch-Reid M, Wilks R. Fast-food and sweetened beverage consumption: association with overweight and high waist circumference in adolescents. Public Health Nutr. 2009;12:1106–14.

122. Fiorito LM, Marini M, Francis LA, Smiciklas-Wright H, Birch LL. Beverage intake of girls at age 5 y predicts adiposity and weight status in childhood and adolescence. Am J Clin Nutr. 2009;90:935–42.

123. Gillis LJ, Bar-Or O. Food Away from Home, Sugar-Sweetened Drink Consumption and Juvenile Obesity. J Am Coll Nutr. 2003;22:539–45.

124. Nguyen S, Choi HK, Lustig RH, Hsu C. Sugar-Sweetened Beverages, Serum Uric Acid, and Blood Pressure in Adolescents. J Pediatr. 2009;154:807–13.

125. Martin-Calvo N, Martínez-González M-A, Bes-Rastrollo M, Gea A, Ochoa MC, Marti A, GENOI Members. Sugar-sweetened carbonated beverage consumption and childhood/adolescent obesity: a case-control study. Public Health Nutr. 2014;17:2185–93.

126. Pollock NK, Bundy V, Kanto W, Davis CL, Bernard PJ, Zhu H, Gutin B, Dong Y. Greater Fructose Consumption Is Associated with Cardiometabolic Risk Markers and Visceral Adiposity in Adolescents. J Nutr. 2012;142:251–7.

127. Kelishadi R, Ardalan G, Gheiratmand R, Gouya MM, Razaghi EM, Delavari A, Majdzadeh R, Heshmat R, Motaghian M, Barekati H, et al. Association of physical activity and dietary behaviours in relation to the body mass index in a national sample of Iranian children and adolescents: CASPIAN Study. Bull World Health Organ. 2007;85:19–26.

128. Bradlee ML, Singer MR, Daniels SR, Moore LL. Eating patterns and lipid levels in older adolescent girls. Nutr Metab Cardiovasc Dis. 2013;23:196–204.

129. Moore LL, Singer MR, Qureshi MM, Bradlee ML. Dairy Intake and Anthropometric Measures of Body Fat among Children and Adolescents in NHANES. J Am Coll Nutr. 2008;27:702–10.

130. Carlson JJ, Eisenmann JC, Norman GJ, Ortiz KA, Young PC. Dietary Fiber and Nutrient Density Are Inversely Associated with the Metabolic Syndrome in US Adolescents. J Am Diet Assoc. 2011;111:1688–95.

131. Davis JN, Alexander KE, Ventura EE, Toledo-Corral CM, Goran MI. Inverse relation between dietary fiber intake and visceral adiposity in overweight Latino youth. Am J Clin Nutr. 2009;90:1160–6.

132. Niinikoski H, Jula A, Viikari J, Rönnemaa T, Heino P, Lagström H, Jokinen E, Simell O. Blood Pressure Is Lower in Children and Adolescents With a Low-Saturated-Fat Diet Since Infancy The Special Turku Coronary Risk Factor Intervention Project. Hypertension. 2009;53:918–24.

133. He FJ, Marrero NM, MacGregor GA. Salt and blood pressure in children and adolescents. J Hum Hypertens. 2007;22:4–11.

134. Yang Q, Zhang Z, Kuklina EV, Fang J, Ayala C, Hong Y, Loustalot F, Dai S, Gunn JP, Tian N, et al. Sodium Intake and Blood Pressure Among US Children and Adolescents. Pediatrics. 2012;130:611–9.

135. Zhu H, Pollock NK, Kotak I, Gutin B, Wang X, Bhagatwala J, Parikh S, Harshfield GA, Dong Y. Dietary Sodium, Adiposity, and Inflammation in Healthy Adolescents. Pediatrics. 2014;133:e635–42.

136. Berkey CS, Rockett HH, Willett WC, Colditz GA. Milk, dairy fat, dietary calcium, and weight gain: A longitudinal study of adolescents. Arch Pediatr Adolesc Med. 2005;159:543–50.

137. Demol S, Yackobovitch-Gavan M, Shalitin S, Nagelberg N, Gillon-Keren M, Phillip M. Low-carbohydrate (low & high-fat) versus high-carbohydrate low-fat diets in the treatment of obesity in adolescents. Acta Pædiatrica. 2009;98:346–51.

138. Ghayour-Mobarhan M, Sahebkar A, Vakili R, Safarian M, Nematy M, Lotfian E, Khorashadizadeh M, Tavallaie S, Dahri M, Ferns G. Investigation of the effect of high dairy diet on body mass index and body fat in overweight and obese children. Indian J Pediatr. 2009;76:1145–50.

139. KANT AK. Indexes of Overall Diet Quality: A Review. J Am Diet Assoc. 1996;96:785–91.

140. Kant AK, Graubard BI. A Comparison of Three Dietary Pattern Indexes for Predicting Biomarkers of Diet and Disease. J Am Coll Nutr. 2005;24:294–303.

141. Panagiotakos DB, Pitsavos C, Stefanadis C. Dietary patterns: A Mediterranean diet score and its relation to clinical and biological markers of cardiovascular disease risk. Nutr Metab Cardiovasc Dis. 2006;16:559–68.

142. Trichopoulos D, Lagiou P. Dietary patterns and mortality. Br J Nutr. 2001;85:133–4.

143. Kennedy ET, Ohls J, Carlson S, Fleming K. The Healthy Eating Index: design and applications. J Am Diet Assoc. 1995;95:1103–8.

144. Ford ES, Mokdad AH, Liu S. Healthy Eating Index and C-reactive protein concentration: findings from the National Health and Nutrition Examination Survey III, 1988–1994. Eur J Clin Nutr. 2004;59:278–83.

145. Guo X, Warden BA, Paeratakul S, Bray GA. Healthy Eating Index and obesity. Eur J Clin Nutr. 2004;58:1580–6.

146. Hurley KM, Oberlander SE, Merry BC, Wrobleski MM, Klassen AC, Black MM. The Healthy Eating Index and Youth Healthy Eating Index Are Unique, Nonredundant

Measures of Diet Quality among Low-Income, African American Adolescents. J Nutr. 2009;139:359–64.

147. Goodwin DK, Knol LL, Eddy JM, Fitzhugh EC, Kendrick OW, Donahue RE. The Relationship between Self-Rated Health Status and the Overall Quality of Dietary Intake of US Adolescents. J Am Diet Assoc. 2006;106:1450–3.

148. Guenther PM, Casavale KO, Reedy J, Kirkpatrick SI, Hiza HAB, Kuczynski KJ, Kahle LL, Krebs-Smith SM. Update of the Healthy Eating Index: HEI-2010. J Acad Nutr Diet. 2013;113:569–80.

149. Guenther PM, Krebs-Smith SM, Reedy J, Britten P, Juan W, Lino M, Carlson A, Hiza HA, Basiotis PP. Healthy Eating Index 2005 Fact Sheet [Internet]. USDA Center for Nutrition Policy and Promotion; 2008 Jun p. 1. Available from: www.cnpp.usda.gov/HealthyEatingIndex.htm

150. Banfield EC, Liu Y, Davis JS, Chang S, Frazier-Wood AC. Poor Adherence to US Dietary Guidelines for Children and Adolescents in the National Health and Nutrition Examination Survey Population. J Acad Nutr Diet. 2016;116:21–7.

151. O'Neil CE, Nicklas TA, Zanovec M, Cho SS, Kleinman R. Consumption of whole grains is associated with improved diet quality and nutrient intake in children and adolescents: the National Health and Nutrition Examination Survey 1999–2004. Public Health Nutr. 2011;14:347–55.

152. De Andrade SC, de Azevedo Barros MB, Carandina L, Goldbaum M, Cesar CLG, Fisberg RM. Dietary Quality Index and Associated Factors among Adolescents of the State of Sao Paulo, Brazil. J Pediatr. 2010;156:456–60.

153. Assumpção D de, Barros MB de A, Fisberg RM, Carandina L, Goldbaum M, Cesar CLG. Diet quality among adolescents: a population-based study in Campinas, Brazil. Rev Bras Epidemiol. 2012;15:605–16.

154. Acar Tek N, Yildiran H, Akbulut G, Bilici S, Koksal E, Gezmen Karadag M, Sanl?er N. Evaluation of dietary quality of adolescents using Healthy Eating Index. Nutr Res Pract. 2011;5:322–8.

155. Nansel TR, Haynie DL, Lipsky LM, Laffel LMB, Mehta SN. Multiple Indicators of Poor Diet Quality in Children and Adolescents with Type 1 Diabetes Are Associated with Higher Body Mass Index Percentile but not Glycemic Control. J Acad Nutr Diet. 2012;112:1728–35.

156. Landy DC, Lipsitz SR, Kurtz JM, Hinkle AS, Constine LS, Adams MJ, Lipshultz SE, Miller TL. Dietary quality, caloric intake, and adiposity of childhood cancer survivors and their siblings: an analysis from the cardiac risk factors in childhood cancer survivors study. Nutr Cancer. 2013;65:547–55.

157. Khalil CB, Johnson-Down L, Egeland GM. Emerging obesity and dietary habits among James Bay Cree youth. Public Health Nutr. 2010;13:1829–37.

158. Harshfield GA, Alpert BS, Willey ES, Somes GW, Murphy JK, Dupaul LM. Race and gender influence ambulatory blood pressure patterns of adolescents. Hypertension. 1989;14:598–603.

159. Luft FC, Miller JZ, Grim CE, Fineberg NS, Christian JC, Daugherty SA, Weinberger MH. Salt sensitivity and resistance of blood pressure. Age and race as factors in physiological responses. Hypertension. 1991;17:I102.

160. Daniels SR, Khoury PR, Morrison JA. The Utility of Body Mass Index as a Measure of Body Fatness in Children and Adolescents: Differences by Race and Gender. Pediatrics. 1997;99:804–7.

161. Wang, Claire Y, Gortmaker, SL, Taveras, EM. Trends and racial/ethnic disparities in severe obesity among US children and adolescents, 1976-2006. Int J Pediatr Obes. 2011;6:12–20.

162. Veugelers PJ, Fitzgerald AL, Johnston E. Dietary Intake and Risk Factors for Poor Diet Quality Among Children in Nova Scotia. Can J Public Health. 2005;96:212–6.

163. Hiza HAB, Casavale KO, Guenther PM, Davis CA. Diet Quality of Americans Differs by Age, Sex, Race/Ethnicity, Income, and Education Level. J Acad Nutr Diet. 2013;113:297–306.

164. Cooke LJ, Wardle J. Age and gender differences in children's food preferences. Br J Nutr. 2005;93:741–6.

165. Larson NI, Perry CL, Story M, Neumark-Sztainer D. Food Preparation by Young Adults Is Associated with Better Diet Quality. J Am Diet Assoc. 2006;106:2001–7.

166. Darmon N, Drewnowski A. Does social class predict diet quality? Am J Clin Nutr. 2008;87:1107–17.

167. Galobardes B, Morabia A, Bernstein MS. Diet and socioeconomic position: does the use of different indicators matter? Int J Epidemiol. 2001;30:334–40.

168. Lallukka T, Laaksonen M, Rahkonen O, Roos E, Lahelma E. Multiple socio-economic circumstances and healthy food habits. Eur J Clin Nutr. 2006;61:701–10.

169. Calkins SD, Blandon AY, Williford AP, Keane SP. Biological, behavioral, and relational levels of resilience in the context of risk for early childhood behavior problems. Dev Psychopathol. 2007;19:675–700.

170. Hollingshead A. Four factor index of social status. New Haven, CT: Yale University; 1975.

171. Merchant AT, Anand SS, Vuksan V, Jacobs R, Davis B, Teo K, Yusuf S, Investigators for the S and S-A. Protein Intake Is Inversely Associated with Abdominal Obesity in a Multi-Ethnic Population. J Nutr. 2005;135:1196–201.

172. Westerterp-Plantenga MS, Lejeune MPGM, Nijs I, van Ooijen M, Kovacs EMR. High protein intake sustains weight maintenance after body weight loss in humans. Int J Obes. 2004;28:57–64.

173. Lejeune MPGM, Kovacs EMR, Westerterp-Plantenga MS. Additional protein intake limits weight regain after weight loss in humans. Br J Nutr. 2005;93:281–9.

174. Halkjær J, Tjønneland A, Thomsen BL, Overvad K, Sørensen TI. Intake of macronutrients as predictors of 5-y changes in waist circumference. Am J Clin Nutr. 2006;84:789–97.

175. Halkjær J, Olsen A, Overvad K, Jakobsen MU, Boeing H, Buijsse B, Palli D, Tognon G, Du H, van der A DL, et al. Intake of total, animal and plant protein and subsequent changes in weight or waist circumference in European men and women: the Diogenes project. Int J Obes. 2011;35:1104–13.

176. Lichtman SW, Pisarska K, Berman ER, Pestone M, Dowling H, Offenbacher E, Weisel H, Heshka S, Matthews DE, Heymsfield SB. Discrepancy between self-reported and actual caloric intake and exercise in obese subjects. N Engl J Med. 1992;327:1893–8.

177. Lissner L, Troiano RP, Midthune D, Heitmann BL, Kipnis V, Subar AF, Potischman N. OPEN about obesity: recovery biomarkers, dietary reporting errors and BMI. Int J Obes. 2007;31:956–61.

178. Macdiarmid J, Blundell J. Assessing dietary intake: Who, what and why of under-reporting. Nutr Res Rev. 1998;11:231–53.

179. Fisher JO, Johnson RK, Lindquist C, Birch LL, Goran MI. Influence of Body Composition on the Accuracy of Reported Energy Intake in Children. Obes Res. 2000;8:597–603.

180. Fulgoni III V, Nicholls J, Reed A, Buckley R, Kafer K, Huth P, Dirienzo D, Miller GD. Dairy Consumption and Related Nutrient Intake in African-American Adults and Children in the United States: Continuing Survey of Food Intakes by Individuals 1994-1996, 1998, and the National Health and Nutrition Examination Survey 1999-2000. J Am Diet Assoc. 2007;107:256–64.

181. Beydoun MA, Gary TL, Caballero BH, Lawrence RS, Cheskin LJ, Wang Y. Ethnic differences in dairy and related nutrient consumption among US adults and their association with obesity, central obesity, and the metabolic syndrome. Am J Clin Nutr. 2008;87:1914–25.

182. King DE, Mainous III AG, Lambourne CA. Trends in Dietary Fiber Intake in the United States, 1999-2008. J Acad Nutr Diet. 2012;112:642–8.

183. Goff DC, Lloyd-Jones DM, Bennett G, Coady S, D'Agostino RB, Gibbons R, Greenland P, Lackland DT, Levy D, O'Donnell CJ, et al. 2013 ACC/AHA Guideline on the Assessment of Cardiovascular Risk A Report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines. Circulation. 2014;129:S49–73.

184. National High Blood Pressure Education Program Working Group on High Blood Pressure in Children and Adolescents. The fourth report on the diagnosis, evaluation, and treatment of high blood pressure in children and adolescents. Pediatrics. 2004;114:555–76.

185. Hajjar I, Kotchen T. Regional Variations of Blood Pressure in the United States Are Associated with Regional Variations in Dietary Intakes: The NHANES-III Data. J Nutr. 2003;133:211–4.

186. Obisesan TO, Vargas CM, Gillum RF. Geographic Variation in Stroke Risk in the United States Region, Urbanization, and Hypertension in the Third National Health and Nutrition Examination Survey. Stroke. 2000;31:19–25.

187. Glazer G. Atherogenic effects of anabolic steroids on serum lipid levels: A literature review. Arch Intern Med. 1991;151:1925–33.

188. Connelly P, Petrasovits A, Stachenko S, MacLean D, Little J, Chockalingam A. Prevalence of high plasma triglyceride combined with low HDL-C levels and its association with smoking, hypertension, obesity, diabetes, sedentariness and LDL-C levels in the Canadian population. Canadian Heart Health Surveys Research Group. Can J Cardiol. 1999;15:428–33.

189. Millán J, Pintó X, Muñoz A, Zúñiga M, Rubiés-Prat J, Pallardo LF, Masana L, Mangas A, Hernández-Mijares A, González-Santos P, et al. Lipoprotein ratios: Physiological significance and clinical usefulness in cardiovascular prevention. Vasc Health Risk Manag. 2009;5:757–65.

190. Kit BK, Kuklina E, Carroll MD, Ostchega Y, Freedman DS, Ogden CL. Prevalence of and trends in dyslipidemia and blood pressure among US children and adolescents, 1999-2012. JAMA Pediatr. 2015;169:272–9.

191. Castelli WP, Anderson K. Selection of Initial Antihypertensive Therapy: New Perspectives on Coronary Heart Disease Risk Factors Provide New InsightsA population at risk: Prevalence of high cholesterol levels in hypertensive patients in the framingham study. Am J Med. 1986;80:23–32.

192. How Is Metabolic Syndrome Diagnosed? - NHLBI, NIH [Internet]. [cited 2016 Apr 24]. Available from: http://www.nhlbi.nih.gov/health/health-topics/topics/ms/diagnosis

193. Maag R, Heath J, Foy A. Fitness and Body Mass Index: Relation to Diabetes, Hypertension and High Cholesterol Among Patients Referred for Symptom Limited Exercise Treadmill Testing. J Am Coll Cardiol. 2016;67:2031–2031.

194. Artero EG, Ruiz JR, Ortega FB, España-Romero V, Vicente-Rodríguez G, Molnar D, Gottrand F, González-Gross M, Breidenassel C, Moreno LA, et al. Muscular and cardiorespiratory fitness are independently associated with metabolic risk in adolescents: the HELENA study. Pediatr Diabetes. 2011;12:704–12.

195. Eisenmann JC, Welk GJ, Ihmels M, Dollman J. Fatness, Fitness, and Cardiovascular Disease Risk Factors in Children and Adolescents: Med Sci Sports Exerc. 2007;39:1251–6.

196. Williams PT. Vigorous Exercise, Fitness and Incident Hypertension, High Cholesterol, and Diabetes. Med Sci Sports Exerc. 2008;40:998–1006.

197. Carnethon MR, Gulati M, Greenland P. PRevalence and cardiovascular disease correlates of low cardiorespiratory fitness in adolescents and adults. JAMA. 2005;294:2981–8.

198. Rahman M, Berenson AB. Accuracy of current body mass index obesity classification for white, black and Hispanic reproductive-age women. Obstet Gynecol. 2010;115:982–8.

199. Burkhauser RV, Cawley J. Beyond BMI: The value of more accurate measures of fatness and obesity in social science research. J Health Econ. 2008;27:519–29.

200. Beller JP, McCartney CR. Cardiovascular risk and combined oral contraceptives: clinical decisions in settings of uncertainty. Am J Obstet Gynecol. 2013;208:39–41.

201. Naz F, Jyoti S, Akhtar N, Afzal M, Siddique YH. Lipid profile of women using oral contraceptive pills. Pak J Biol Sci PJBS. 2012;15:947–50.

202. Mishra S, Xu J, Agarwal U, Gonzales J, Levin S, Barnard ND. A multicenter randomized controlled trial of a plant-based nutrition program to reduce body weight and cardiovascular risk in the corporate setting: the GEICO study. Eur J Clin Nutr. 2013;67:718–24.

203. Blackburn G. Effect of Degree of Weight Loss on Health Benefits. Obes Res. 1995;3:211s – 216s.

204. Dj G. Beneficial health effects of modest weight loss. Int J Obes Relat Metab Disord J Int Assoc Study Obes. 1992;16:397–415.

205. Sesso HD, Stampfer MJ, Rosner B, Hennekens CH, Gaziano JM, Manson JE, Glynn RJ. Systolic and Diastolic Blood Pressure, Pulse Pressure, and Mean Arterial Pressure as Predictors of Cardiovascular Disease Risk in Men. Hypertension. 2000;36:801–7.

206. Gavish B, Ben-Dov IZ, Bursztyn M. Linear relationship between systolic and diastolic blood pressure monitored over 24 h: assessment and correlates: J Hypertens. 2008;26:199–209.

207. Castelli WP, Doyle JT, Gordon T, Hames CG, Hjortland MC, Hulley SB, Kagan A, Zukel WJ. HDL cholesterol and other lipids in coronary heart disease. The cooperative lipoprotein phenotyping study. Circulation. 1977;55:767–72.

208. Moore LL, Singer MR, Bradlee ML, Djouss L, Proctor MH, Cupples LA, Ellison RC. Intake of Fruits, Vegetables, and Dairy Products in Early Childhood and Subsequent Blood Pressure Change: Epidemiology. 2005;16:4–11.

209. Miller PE, Mitchell DC, Harala PL, Pettit JM, Smiciklas-Wright H, Hartman TJ. Development and evaluation of a method for calculating the Healthy Eating Index-2005 using the Nutrition Data System for Research. Public Health Nutr. 2011;14:306–13.

APPENDIX A

HEI-2010 SCORE CALCULATION METHODOLOGY

Diet quality, as expressed as HEI-2010, reflects an average of the participant intake over all 3 days of diet recall data. To calculate HEI-2010 scores for each participant, output files 1 (nutrient data at the component/ingredient level), 4 (nutrient data at the daily totals level), and 9 (food group serving counts at the daily totals level) from the NDSR 2013 software were utilized.

Food group serving counts from output file 9 were used to calculate HEI-2010 component scores for total fruit, whole fruit, total vegetables, greens and beans, whole grains, dairy, total protein foods, seafood and plant proteins, and refined grains. Serving counts were converted to ounces for whole grains, total protein foods, seafood and plant proteins, and refined grains. Serving counts for total fruit, whole fruit, total vegetables, greens and beans, and dairy were converted to cups.

The total fruit component of the HEI-2010 includes all fruit listed in the MyPyramid Equivalents Database. From NDSR output file 9, all fruit groups were summed to generate a total fruit amount. The whole fruit component of the HEI-2010 includes all fruits from the total fruit component, excluding 100% fruit juice. In NDSR, all fruit groups, excluding juices, were summed to generate a whole fruit amount. Similarly, total vegetables for the HEI-2010 include all vegetables listed in the MyPyramid Equivalents database. As such, all vegetable groups from the NDSR output file 9 were summed to generate a total vegetables amount. The greens and beans

component is considered the total of all dark green vegetables, dried beans, and peas. The dark green vegetables group and legumes group from the NDSR output was summed to determine a greens and beans amount.

The HEI-2010 whole grains component includes all grains containing the entire grain kernel, which applies to whole wheat flour products, brown rice, and a number of unrefined grains, such as oatmeal, quinoa, barley, etc. NDSR output file 9 includes three groups for grains: whole grains, some whole grains, and refined grains. In order to account for the whole grain portion of the some whole grains group, we estimated that all some whole grains are approximately 50% whole grain(209). To compute the whole grains amount, we summed the whole grains group with half the some whole grains group. For the HEI-2010 refined grains component, the some whole grains group from NDSR output was treated identically. Refined grains, as defined by the HEI-2010, include all grains containing less than the entire kernel. To calculate a refined grains amount, the refined grains group was summed with half the some whole grains group.

The HEI-2010 defines the dairy component as products produced from cow's milk, goat's milk, and fortified soy beverages. While whole fat and reduced fat milk products are both included, those foods made predominantly from milk fat, such as butter, cream, ice cream, and sour cream, are excluded from this component. From NDSR output file 9, all milk, yogurt, and cheese groups were summed to generate a dairy amount, while dairy-based desserts, creams, and dairy-based supplement groups were excluded.

The HEI-2010 total protein foods component includes all meat, poultry, fish, eggs, nuts, legumes, and soy-based meat substitutes such as tofu. The corresponding groups from NDSR output file 9 were summed to generate a total protein foods amount. Seafood and plant proteins are defined by the HEI-2010 as any seafood, nuts and seeds, and soy products, excluding soy beverages. Thus, all fish, shellfish, nuts and seeds, and meat alternatives groups from the NDSR output were summed to generate a seafood and plant proteins amount. Use of beans and peas to count toward the total protein foods component only occurs if the maximum (2.5 oz. equivalents per 1,000 kcal) score is not reached from other protein foods. When beans and peas are counted toward total protein foods, they are only counted up to the threshold for the maximum score. Any beans and peas counted in the total protein foods component are not counted toward the total vegetables or greens and beans components. Any beans and peas intake remaining after the maximum score for the total protein foods component is reached will count toward the total vegetables and greens and peas components. Those beans and peas counted for the total protein foods component are also counted toward the seafood and plant proteins component.

Nutrient data at the daily totals level from NDSR output file 4 was utilized to determine scores for the fatty acids and sodium components. The fatty acids HEI-2010 component is defined as the sum of dietary polyunsaturated fatty acids and monounsaturated fatty acids, then divided by saturated fatty acids. NDSR output file 4 generated total daily intake of these three fatty acid groups, so we used those groups to

calculate the fatty acids component as above. The sodium component of the HEI-2010 is simply the daily intake of sodium. NDSR output file 4 provided this value directly.

Nutrient data at the daily totals level from NDSR output file 4 and at the component/ingredient level from NDSR output file 1 was used to determine the empty calories component score. Empty calories are defined by the HEI-2010 as calories from solid fats, alcohol, and added sugars. NDSR output file 4 provides a total for solid fats in grams. NDSR output file 4 also contains an added sugars group, defined very similarly to the HEI-2010 definition: sugar used in prepared foods, processed foods, and added separately to foods. To determine the alcohol aspect of the empty calories component, the grams of ethanol from NDSR output file 4 was used first. If intake does not exceed 13 grams per 1,000 kcal, calories from alcohol are not considered in the empty calories component. Otherwise, grams ethanol represented one aspect of the empty calories component. NDSR output file 1 was used to identify the other energy-contributing macronutrients present in the alcoholic beverages. Carbohydrate (in grams) from all alcoholic beverages was summed, excluding sugars. Protein (in grams) from all alcoholic beverages was summed. Lastly, fat (in grams) from alcoholic beverages, excluding trans and saturated fats, was summed. These macronutrients were summed to represent all energy derived from the alcoholic beverages. Sugar was omitted, as it was included in the added sugars amount. Trans and saturated fats were omitted here, as they were included in the solid fats amount.

To derive a score for the empty calories component, all data in grams must be converted to energy. Added sugars, carbohydrates from alcoholic beverages, and proteins from alcoholic beverages were multiplied by 4 kcal/gram. Solid fats and fats from alcoholic beverages were multiplied by 9 kcal/gram. Ethanol was multiplied by 7 kcal/gram. Energy from the above six was summed, then divided by total energy, and multiplied by 100 percent. This percentage was used to score the HEI-2010 empty calories component as below. Total energy consumed from NDSR output file 4 was used for total energy.

[(50% - average 3 day %energy from empty calories)/ 31] x 20 =

Participant Empty Calories Component Score

50% represents the standard for a minimum score in this category, while 31 is the difference between the standard for a minimum and maximum score.

All adequacy components of the HEI-2010 that are converted to cup equivalents (total fruit, whole fruit, total vegetables, greens and beans, dairy) were scored as below:

(3 day average cup equivalents consumed/ 3 day average for energy) x 1000 = Participant Standard

(Participant Standard/ Component Standard for Maximum Score) x maximum points possible = Component score

All adequacy components of the HEI-2010 that are converted to ounce equivalents (whole grains, total protein foods, seafood and plant proteins) were scored in the same manner as above, replacing cup equivalents for ounce equivalents.

The refined grains component of the HEI-2010 was scored as below:

(3 day average ounce equivalents consumed/ 3 day average for energy) x 1000 = Participant Refined Grain Standard

[(4.3 – Participant Refined Grain Standard)/ 2.5] x 10 =

Refined Grains Component Score

4.3 represents the standard for a minimum score in this category, while 2.5 is the difference between the standard for a minimum and maximum score.

The HEI-2010 sodium component was scored similarly:

(3 day average gram sodium consumed/ 3 day average for energy) x 1000 = Participant Sodium Standard

[(2 – Participant Sodium Standard)/ 0.9] x 10 = Sodium Component Score

2 represents the standard for a minimum score in this category, while 0.9 is the difference between the standard for a minimum and maximum score.

Finally, the HEI-2010 fatty acids component was scored as below:

(3 day average grams polyunsaturated fatty acids + 3 day average grams monounsaturated fatty acids)/ 3 day average grams saturated fatty acids = Participant Fatty Acids Standard

[(Participant Fatty Acids Standard – 1.2)/ 1.3] x 10 = Fatty Acids Component Score

1.3 represents the standard for a minimum score in this category, while 1.2 is the difference between the standard for a minimum and maximum score.

In each case above, if the calculated score exceeds the maximum points for the component, the maximum points for that component replaced the score.

Dietary Components

In order to isolate the data for specific dietary components and food groups, output files 4 (nutrient data at the daily totals level) and 9 (food group serving counts at the daily totals level) from the NDSR 2013 software were utilized.

Total energy, total fat, saturated fat, omega-3 fatty acid, fiber, sodium, potassium, calcium, and magnesium intake were provided in output file 4. The intakes of each category were summed for the three days of diet data, then divided by three to derive an average daily intake. Total and saturated fat were additionally multiplied by 9 kcal per gram, then divided by the 3 day average for energy, and multiplied by 100 to derive a percentage. These average daily intakes were utilized in data analyses.

Refined grain, empty calorie, whole grain, fruit, vegetable, and legume intakes were determined in the same method as above, using NDSR output file 9. The average cup equivalents consumed for each were used for each group, as opposed to converting to an HEI-2010 component score. The legumes intake was determined solely from the average of the three days' diet recall data for the legumes group in NDSR output file 9.